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MAURICE WILLIAM AND SUN YAT-SEN

"The meeting of two minds and of two civilizations: American and Chinese"

The dean of an eastern dental school, whose efforts to discredit dentists and dentistry have become notorious, has recently circulated a "memorandum" derisively calling attention to a superficial review of William's recent book on "*Sun Yat-sen versus Communism*" (1932). In this "review," a so-called "authoritative writer on China," admitting that the philosophy and phraseology in William's "*Social Interpretation of History*" (1921) were freely used by Sun Yat-sen in the formulation of the Chinese nationalist program, states, however, that this fact is unimportant because Sun Yat-sen has no influence on conditions or events in China. Such "authoritative" comment is sheer nonsense. Dentists who appreciate the great significance of William's remarkable relationship to the development and statement of Sun Yat-sen's national policies, and who—unlike the dean aforesaid—acclaim that relationship as inspiring evidence that the practice of dentistry is not incompatible with intellectual and social achievement of the highest order, will be glad to read the following statements in recent books by men of high repute who are widely known as responsible and scholarly observers in Chinese affairs:

By George E. Sokolsky: "The Tinder Box of Asia," pp. 329–32; July, 1932. (Mr. Sokolsky, the New York Times expert on the Far East—having lived in China for many years—is an intimate, personal friend of most of the leaders in the Nationalist Government at Nanking.)—"Dr. Sun. . . . . began a series of lectures which laid down definitely the principles for which the Nationalists were striving. These lectures are known as the *San Min Chu I*—the Three Principles of the People. . . . . After Dr. Sun's death these lectures became the bible of nationalism. . . . his basic social and economic doctrine was influenced by *The Social Interpretation of History* by an American, Maurice William. This book had as its sub-title "A Refutation of the Marxian Economic Interpretation of History." Dr. Sun mentioned William by name and quoted him extensively. . . . . *In death the Doctor [Sun] was a greater figure than he had ever been while alive* [although while *alive* he was the successful leader of a national revolution]. . . *he has become the national saint.*" [Italic not in the original.]

By Paschal M. D'Elia, S. J.: "The Triple Demism of Sun Yat-sen," pp. 421 and 543–44; 1931. (An original translation from the Chinese into English of Dr. Sun's San Min Chu I, or the Three Principles of the People. Father D'Elia is a member of the Sinological Bureau, Shanghai, China.)—"Dr. Sun drew his best arguments to oppose the doctrine of Marx from Dr. Maurice William's '*The Social Interpretation of History*'. . . . . Since the Nationalists are in power, scarcely a day passes by without an *official edict of the Government adding its stone to the grand edifice of the future nation, a nation to be built anew entirely upon the rock foundation of the Triple Demism* [*Dr. Sun's San Min Chu I, or the Three Principles of the People*]. If China 'awakens'—so speak the Chinese themselves—if she is convinced that she must develop her industries, her education, her administration, her Government—in a word, everything—*she believes that all this is impossible unless it is based on the Triple Demism. The password, the touchstone of everybody and of everything is in this 'ism,' and in this 'ism' alone.* When there is question of incorporating into the Party those who yesterday were enemies, or of planning reforms and mapping out the future, hardly any other question is asked but: "Are you in favor of or opposed to the Triple Demism?"; "Does that law, that plan conform to the Triple Demism?" Things have gone so far that sentences of death or of life imprisonment have been contemplated against all anti-revolutionists, meaning thereby all those who would be bold enough to hold a doctrine opposed to the Triple Demism. *Foreigners who witness a fact of this kind cannot understand it; they cannot go that far. But they are forced to bow before it and to admit it even in spite of themselves.* Sun Yat-sen's book has been broadcasted all over the country. *It is meant to be the code of the new generations.* It has to bring forth a new nation. *In the mind of the present leaders of China this book must be made a compulsory text for all the schools, even mission schools.* Mention is even made of one school for girls which used no other textbook nor program of study but the Triple Demism. At the end of the school-year students must pass a special examination upon this new topic. *In the tests for government office the candidate must, first of all, show that he has mastered the Demist doctrine.*" [Italic not in the original.]

The report of the League of Nations' "Commission on the Manchurian Dispute," as published in the daily press on Oct. 3, 1932, contained, among its ten stated "conditions of a satisfactory solution" and as a "*final requisite*," the following recommendation: "temporary international coöperation in the internal reconstruction of China, *as suggested by the late Dr. Sun Yat-sen.*" [Italic not in the original.] Here we see an extension of Sun Yat-sen's living influence from China *into the deliberations of the League of Nations.*

Every dentist who appreciates *the spiritual and intellectual importance, for his profession,* of constructive public service *by dentists* will wish to study William's new book, which is an important contribution to the solution of current international problems, and of economic difficulties in the United States. Laymen should be shown this book as the work of a practising dentist. *Publisher:* Williams and Wilkins Co., Mt. Royal and Guilford Aves., Baltimore, Md.—*William J. Gies, Columbia University, October 10, 1932.*

PROSTHESIS OF THE MOUTH AND FACE

A Symposium[1]

V. H. KAZANJIAN, D.M.D., M.D., F.A.C.S., *Boston, Mass.*; ARTHUR T. ROWE, D.D.S., *New York City*; AND HARRY A. YOUNG, D.D.S., *New York City*

CONTENTS

I. Modern accomplishments in dental and facial prosthesis. *V. H. Kazanjian, D.M.D., M.D., F.A.C.S., Professor of Clinical Oral Surgery, Harvard University, Boston, Mass.* 651
II. Simple prosthesis. *Arthur T. Rowe, D.D.S., Professor of Dentistry, Columbia University, New York City* 670
III. Cleft-palate prosthesis at the Columbia University Dental School Clinic. *Harry A. Young, D.D.S., Assistant Professor of Dentistry, Columbia University, New York City* 675
IV. *Discussion:* Drs. I. W. Voorhees, I. J. Sperber, H. S. Dunning, V. H. Sears, Alfred Michaelis, and V. H. Kazanjian 687

I. MODERN ACCOMPLISHMENTS IN DENTAL AND FACIAL PROSTHESIS

V. H. Kazanjian, D.M.D., M.D., F.A.C.S., Harvard University, Boston, Mass.

Since the sixteenth century, prosthetic restorations have been made to correct defects of the orbit, nose, ear, lower portions of the face, lips, palate, mandible, chin, and larynx. Early records show modest efforts to close perforations of the palate due to syphilitic necrosis. Later, treatment of more complicated deformities was gradually developed. In the dental literature there are references on the subject bearing a long list of distinguished names, such as Ambroise Paré (1541), Pierre Fauchard (1728), Bourdet, Suerson, Delabarre (1820), Hullihan, Kingsley, Claude Martin, and others. These pioneers developed prosthesis to such a high degree that a reservoir of knowledge was available when the excessive demands of the World War suddenly came upon us. Since then the development of surgical prosthesis has been very closely linked to general progress in dentistry.

[1] Proceedings of the American Stomatological Association, Columbia University Club, New York City, November 10, 1931.

Facial and dental defects are caused by congenital malformations, such as cleft palates and secondary deformities from unsuccessful operations on cleft palates; by the destructive effects of diseases, such as osteomyelitis, syphilis, benign or malignant tumors; and by trauma, especially gun-shot wounds. During the early era of surgical prosthesis, the dentist was called upon to make restorations of long-established deformities resulting from treatment where no attempt had been made to minimize inevitable distortion. For example, the patient underwent an operation for the removal of a tumor, and the surgeon had no means to prevent scar formation or contraction of the tissues, with the result that artificial restoration often encountered unusual difficulties owing to uncontrollable conditions of the healing process. In 1875 Claude Martin was the first, I believe, to conceive the idea of immediate prosthesis. With great enthusiasm and daring he devised methods and suitable appliances for such purposes. His procedure involved immediate introduction of a restoration after operation. Such an appliance was made approximately the same size and shape as the tissue excised from the maxilla or mandible. Although we consider most of his appliances complicated and unsuitable for present-day surgical technique, he deserves great credit for his pioneer work in this field. From time immemorial deformities of the face and jaws have also been treated by surgery. Indeed, plastic surgery is an ancient art that has been practised for thousands of years. Over two thousand years ago the Hindus developed methods, among other difficult plastic feats, for the repair of nasal defects that even today are considered good operative procedure in rhinoplasty. The literature of plastic surgery contains the names of many illustrious surgeons dating as far back as Celsus (25 B.C.)—and, in the succeeding centuries, Galen (131–201 A.D.), Paul of Aegina (630 A.D.), Branca (1442), Tagliacozzi (1546), Nelaton and Zeis (1900), up to the pre-war surgeons, Morestin, Lexer, and others.

These two methods of restoration, prosthetic and plastic, were developed independently over a long period of years. Surgeons, unfamiliar with the possibilities of prosthesis, depended solely upon their own resources; and, as a result in cases where destructive processes had extended to the supporting structures of the face, the methods employed were insufficient. Likewise dentists, unfamiliar with sur-

gical methods of restoration, used all their ingenuity in attempts to overcome difficulties by the use of appliances. While many excellent prosthetic restorations have been made to meet all types of conditions, we know that in selected cases surgery would have either made prosthesis unnecessary or greatly simplified its construction. The big step forward in this generation has been the realization that these two phases of the healing art are inseparably related, and that the future of restorative work, just as in the field of prevention, lies in the closest coöperation between medicine and dentistry. The surgeon can no longer ignore the dental art, nor can the dentist achieve entirely satisfactory results without the sympathy and support of the surgeon.

Present-day restoration of the face, involving both surgery and dentistry, falls readily into four classes: orbital, nasal, maxillary, and mandibular. I shall discuss briefly modern methods and technique in each of these four aspects, and reproduce a few case histories for illustration.

Restoration of the orbit

Enucleation of the eye is quite commonly performed by ophthalmic surgeons, and the related problem of prosthesis belongs in their field. But complete removal of orbital contents, including excision of eyelids and surrounding tissues, often becomes necessary for the removal of orbital tumors, or of malignant tumors of the nasal sinuses that have invaded the orbital cavity. Naturally such an operation leaves a large exposed cavity in the face; and while a plastic operation may be considered, such a step is not always advisable, because often a fair cosmetic result can be obtained by prosthetic restoration. Furthermore, a prosthetic restoration has the additional advantage of keeping the cavity free from annoying crusting, and gives the surgeon an opportunity for frequent inspection and treatment in the event of recurrence of the disease.

In general the methods for constructing an orbital restoration fall into two classes. The first comprises a shell-like cover over the eye socket, made of vulcanite or metal and modelled according to the contour of the face. Inside of this shell, an artificial eye is inserted, and retention is obtained by fastening the whole appliance to the frame of spectacles. The second method consists of utilizing the entire

FIG. 1

FIG. 2

FIG. 1. BASAL-CELL CARCINOMA BEFORE OPERATION
FIG. 2. PATIENT AFTER OPERATION

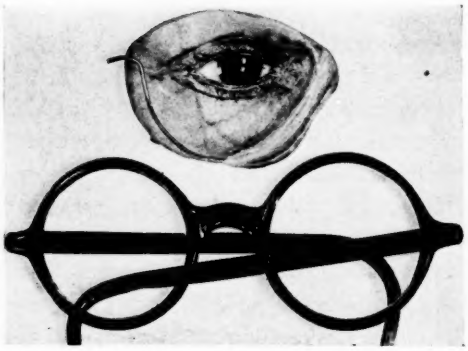


FIG. 3a

FIG. 3b

FIG. 3a. ARTIFICIAL ORBIT MADE OF VULCANITE RUBBER
FIG. 3b. INNER SURFACE OF PROSTHESIS, AND RELATION TO FRAME OF SPECTACLES

orbital cavity for the purpose of retention. The restoration not only covers the defect, but also extends into the cavity, and is in contact with the floor and lateral walls of the orbit. I have always preferred the second method as providing more efficient retention, and also affording greater comfort to the patient.

FIG. 5. PATIENT WITH PROSTHESIS IN POSITION

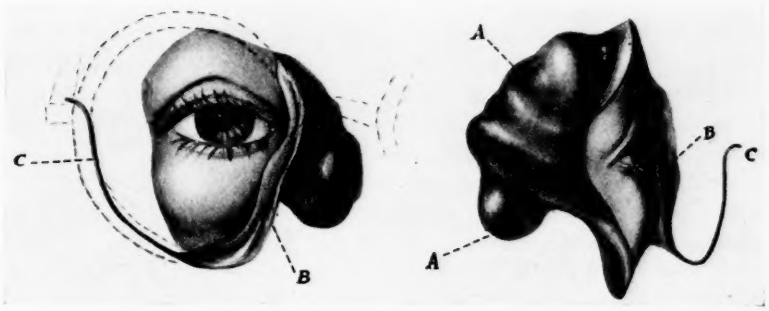


FIG. 4. DIAGRAM OF ARTIFICIAL ORBIT, SHOWING FURTHER DETAILS OF RETENTION

Case I: Miss E. B.—Patient came to Tumor Clinic of Massachusetts General Hospital, *Dec. 8, 1930.* Had basal-cell carcinoma of orbit and lower lid; twelve years duration. Lesion had been growing steadily in spite of x-ray therapy. Examination showed malignancy largely destroyed lower lid and lower part of eyeball. Biopsy report: basal-cell carcinoma; patient advised to undergo operation. *Dec. 11:* operation performed at Massachusetts Eye and Ear Infirmary; right orbital contents removed, including entire lower lid and part of upper lid. Whole surface of wound cauterized with diathermy (*fig. 1*). *Sep. 24, 1931:* patient referred to Harvard Dental School for orbital prosthesis (*fig. 2*). Wound had healed satisfactorily; no evidence of recurrence. At Department of Surgical

Prosthesis artificial restoration made, consisting of vulcanite base covering entire orbital cavity, with extension downward toward maxillary sinus. Over this base, artificial eyelids were molded with clay approximating normal eyelids as nearly as possible; later transferred to vulcanite. Prosthetic eye put in to match left eye; artificial eyelashes (horsehair) inserted. Surface of eyelids painted with celluloid paints to match skin (*fig. 3*). In prosthetic restorations of this type means for retention of prosthesis very important. In this case retention acquired, *first*, by extending base to cover entire orbital cavity (*fig. 4, a*), part of which extended to maxillary antrum, as there was opening leading to sinus; *secondly*, special groove made along nasal side of prosthesis to fit frame of spectacles (*fig. 4, b*). In addition, spring wire extended from lower end of groove behind lower rim of spectacles to hinge of bow (*fig. 4, c*). Patient used restoration very comfortably, general appearance very satisfactory (*fig. 5*).

Case II: T. S., age 59.—Right eye removed by Dr. C. A. Porter, *Oct., 1928*. Examination, *Dec. 29, 1930*: no recurrence of malignancy (*figs. 6* and *7*); wound had healed, but constant crusting of secretions bothered patient; entire orbital contents removed, including upper and lower eyelids; medial wall of orbit removed up to side of nose, thus exposing nasal cavity as well. Orbital prosthesis made by same technique as for Case 1. Retention of prosthesis acquired by covering floor, external wall, and roof of orbit with vulcanite base, but leaving nasal side open (*fig. 8, a*); special groove also made along base of nose to receive rim of spectacles (*fig. 8, b*). Patient using this restoration for about a year; cavity of orbit free from crusting; healthier in appearance (*figs. 9* and *10*).

Restoration of the nose

Excepting cases of extensive damage from gunshot wounds, defects of the nose from ordinary accidents hardly cause enough destruction to warrant artificial restoration. Invariably these cases are treated by surgery. Here also gross deformities are mostly due to carcinoma, although occasionally to lupus. Sometimes, of course, tissues adjacent to the nose are involved, complicating the technique of restoration. Rhinoplasty has made great advances in the last few years. Nasal restorations are now frequently performed by surgery that formerly were considered impracticable. But in certain types of these deformities, surgery has its limitations, not so much because adequate surgical technique is lacking but rather because the picture is obscured by other factors over which we have no control, such as the physical

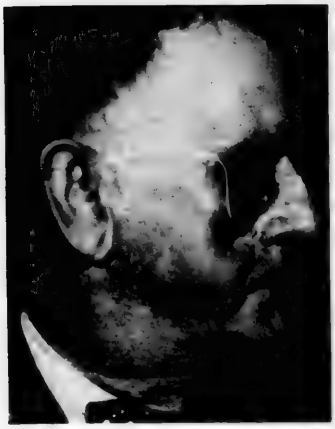


FIG. 6

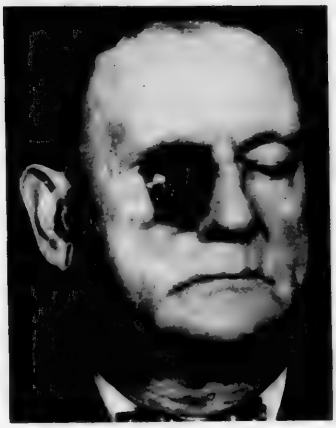


FIG. 7

FIGS. 6 AND 7. DEFORMITY INVOLVING RIGHT ORBIT, PART OF FACE AND SIDE OF NOSE

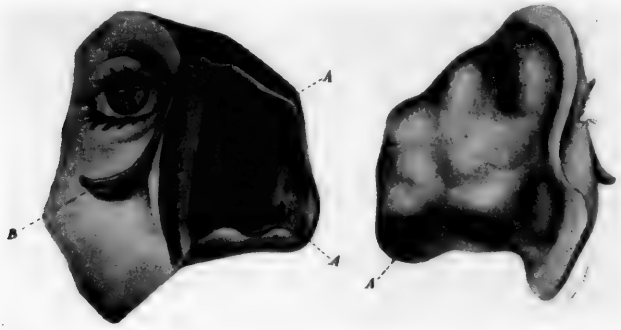


FIG. 8. DIAGRAM SHOWING VULCANITE RESTORATION, WITH VARIOUS DETAILS FOR RETENTION

FIG. 9

FIG. 10

FIGS. 9 AND 10. PROSTHESIS IN POSITION

condition of the patient, peculiarities of the disease that caused the deformity, and the refusal of the patient to undergo surgical treatment. Extensive reconstruction of the nose often requires a series of operations. Patients undergoing such operations should have a fair degree of health and vitality. Most patients having gross nasal deformities are past middle age, and therefore weakened to some extent by the disease that caused the deformity. In cases of carcinoma, besides the factor of age, the disease is treacherous and progressive by nature. It is not uncommon to see beautifully performed plastic operations end in disaster in a recurrence of the malignancy. While appreciating the great possibilities of surgery, one must therefore consider the age and physical condition of the patient, and the peculiarities of the disease. We are thus often compelled to choose prosthesis as the best means for the correction of nasal and facial deformities, realizing that while artificial restorations have great cosmetic possibilities, they are after all artificial and "false," and carry with them an element of repulsion.

In a previous paper,[2] I outlined in considerable detail a method of nasal prosthesis whereby many cases have been treated with success. I shall not describe each step, but a few fundamental points will be emphasized. Next to molding a suitable shape of nose to harmonize with the contour of the surrounding tissues, the problem of retention is most important. To give the maximum retention, the following methods, either singly or in combination, may be utilized. The base of the artificial nose should rest upon as much tissue as is compatible with its form. There should be a horizontal extension from the base of the appliance to the floor of the nose, to prevent it from sliding downward. The bridge and the nasal sides of the spectacles used for retention should fit accurately upon the nose in definite grooves, and the greatest pressure from the spectacles should be at their lowest point of contact half-way down the side of the nose. If there is a perforation of the palate to the nasal floor, an attachment should be provided to connect the artificial nose to the upper denture. This feature, combined with the use of spectacles and a good fitting base, will insure the maximum retention. Painting the facial restoration

[2] Kazanjian: *Journal of the American Medical Association*, 1925, lxxxiv, p. 177; also Nichols' Prosthetic Dentistry, p. 513.

is a great problem. While the skilled artist can imitate more or less the color of the face, the color of the skin is so sensitive to variation of temperature, as well as variations under different lights, that no coloring matter has yet been devised that will accommodate this condition. This is the greatest handicap in artificial facial restorations. Another method to approximate the appearance of normal tissue, advocated by Pont and others, involves the use of gelatin. The product imitates the softness of the natural nose and somewhat the texture of the skin, but it must be renewed every week or so. This disadvantage may be partly offset, however, by having the patient pour the gelatin composition into a mold, thus preparing the prosthesis himself. Dr. G. G. Bercowitsch advocates a formula for gelatin which is different from the formula used by Pont.[3]

Case III: E. S.; age 73.—Patient seen *Feb. 20, 1939.* Examination showed extensive squamous-cell carcinoma involving greater part of nose; had already invaded septum and mucous membrane of nose (*fig. 11*). *Apr. 3:* patient operated under general anesthesia. With electro-surgical knife, entire diseased tissue excised with good margin of healthy tissue, involving, in addition to bony and cartilaginous structures, anterior part of septum and upper half of upper lip. Entire surface of wound cauterized with diathermy. *Sep. 9:* wound of face completely healed; upper lip, however, consisted of thin strip of tissue not over 1 cm. wide, and bound down by adherent scars (*figs. 12* and *13*). Operated to remedy condition. Under local anesthesia, upper lip elongated to proper size by making oblique incision throughout entire thickness of lip, and overlapping flaps until proper size acquired (*fig. 14*). *Oct. 2:* artificial nose constructed with vulcanite rubber (*fig. 15*). Main features of prosthesis: (*a*) inner surface (*fig. 15*, *a*, base) fitted over face accurately and covered as much space as

[3] "Our clinical recipe is as follows: We soak 10 gm. of gelatin in 50 cc. of glycerine; the surplus, about 33 cc. is poured out, after which the gelatin is carefully pressed out in a towel, heated in a double boiler, and the following added:

Glycerini	15.0 cc.
Aq. destil	7.5 cc.
Spir. vini	5.0 cc.
Ac. salicyl	0.5 gm.
Kal. bichrom	iij gtt.

With the above modifications in the methods of the foreign authors we obtained a far greater stability of the prosthesis." Bercowitsch: *Dental Cosmos*, 1928, lxx, p. 167.

FIG. 11 FIG. 14 FIG. 16

FIG. 11. SQUAMOUS-CELL CARCINOMA, INVOLVING NOSE AND ADJACENT TISSUES
FIG. 14. PATIENT PRIOR TO CONSTRUCTION OF PROSTHESIS
FIG. 16. PROSTHESIS IN POSITION

FIG. 12a FIG. 12b FIG. 13

FIG. 12. DIAGRAM SHOWING INCISION LINES FOR REPAIR OF UPPER LIP
FIG. 13. DIAGRAM SHOWING POSITION OF FLAPS FOR ELONGATION OF UPPER LIP

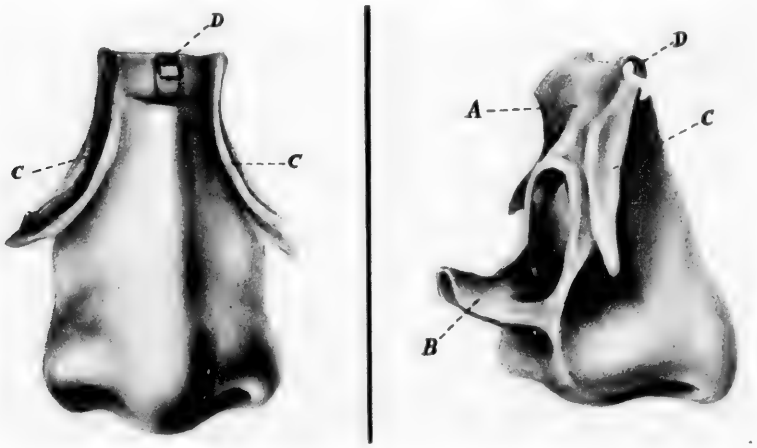


FIG. 15. DIAGRAM OF NASAL PROSTHESIS

FIG. 17 FIG. 18 FIG. 19

FIG. 17. PATIENT WITH CARCINOMA OF RIGHT HALF OF NOSE
FIG. 18. IMMEDIATE SURGICAL REPAIR, FOLLOWING FIRST OPERATION FOR REMOVAL OF CARCINOMA
FIG. 19. DEFORMITY AFTER SECOND OPERATION

FIG. 20a FIG. 20b

FIG. 20. PROSTHESIS IN POSITION

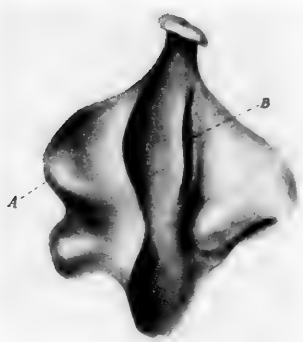


FIG. 21a FIG. 21b

FIG. 21. DIAGRAM OF PROSTHESIS

consistent to shape of nose; (*b*) prolongations of restoration (*fig. 15, b*) rested upon floor of nose, purpose being to prevent artificial nose from sliding down upon lip; (*c*) lateral grooves as well as metal clasp over bridge (*fig. 15, c–d*) fitted accurately to bridge and rims of spectacles, so that when nose is worn, there is even pressure from spectacles to upper half against tissues of face (*fig. 16*). Nose painted with celluloid paint to match color of skin. Patient seen as late as *Nov., 1931*; wearing restoration comfortably.

Case IV: Mr. C.; combined defect of side of face and half of nose.—Patient operated at Massachusetts Eye and Ear Infirmary, in *1925*, for small carcinoma of lip and side of nose. Plastic operation performed immediately afterward for repair of nose (*figs. 17* and *18*). Patient not seen again until *1930*, although follow-up cards repeatedly sent from hospital asking him to report for observation. *Oct., 1930:* examination showed extensive recurrence of carcinoma involving entire right side of nose, naso-antral wall, maxillary sinus, and side of face. Operation performed with electro-surgical knife, when side of nose and part of face tissues excised, maxillary sinus cleaned, and entire naso-antral wall removed; right upper and lower eyelids sutured together to prevent possible distortion of lower lid from resulting scars, and to protect eye from exposure (*fig. 19*). *May, 1931:* patient supplied with restoration at Harvard Dental School (*fig. 20*), consisting of one-half nose and extension covering opening of face, made of vulcanite rubber having prolongations to rest upon floor of nose and maxillary sinus (*fig. 21, a*) and groove adapted to contour of rim and bridge of spectacles (*fig. 21, b*). Advantages of this method, in addition to appearance: permits frequent inspection of wound for possible recurrence of disease, and also preserves humidity of nasal cavity.

Restoration of the upper jaw

In maxillary restoration, prosthetic procedure can be utilized with great effectiveness. Destruction of the palate may vary in size and shape, ranging from small perforations to total obliteration of the hard palate and even of the soft palate. The case may be complicated by various adhesions, trismus of the jaw, and absence of suitable teeth for retention. Surgical repair is necessary for cases of small perforation; also for adhesions and scars, to facilitate construction of the artificial denture. In this respect, one important recent advance has been a realization that the introduction of prosthesis immediately after an operation for the removal of a tumor of the upper jaw not only makes the patient more comfortable while con-

valescing but also prevents many avoidable deformities while the tissues are healing. Not infrequently the surgeon is called upon to do a skin graft within the buccal cavity in order to prepare the field for prosthesis.

With the denture, the greatest difficulty of construction lies in the means for retention. Generally we depend first upon the remaining teeth; secondly, upon the remaining part of the hard palate and alveolar process to provide a base for stability; third, upon the borders of the perforation and different surfaces of the nasal cavity by means of extensions; finally, upon different types of spiral springs. Restorations for extensive deformities of the jaw are naturally quite bulky, and in some instances are constructed in more than one section. So far there have not been any hard and fast rules for the general design of these appliances excepting the abovementioned basic principles of retention. There is a tendency toward the simplification of appliances. This is made possible partly because surgery is called upon to prepare the mouth for their adaptation.

Case V: Mr. T. K.; age 33.—Patient reported for first time in *Sep., 1929.* Gave history of operation on right upper jaw, in 1925, for endothelioma, followed with x-ray treatment. Examination: local swelling on right side of maxilla extending toward nose and pushing palate downward. X-ray picture showed involvement of right upper jaw but not of antrum. *Sep. 7, 1929:* under ether, greater part of right side of upper jaw removed, including part of palate and entire naso-antral wall; antrum exposed, and all contents removed. Patient made good recovery. Denture made to cover defect of palate and patient got along comfortably until *May, 1930,* when he noticed soreness on palate directly below septum. Examination: increased swelling and possible recurrence of tumor on floor of left side of nose. Pathological report: "undifferentiated type of sarcoma." *May 27:* two platinum needles each containing 10 mg. of radium introduced directly into tumor; total radiation, 2000 mc hours. Tumor tissue totally disappeared (*fig. 22*). Later, new appliance made, and retained in position by clasps (18-gauge) around right third molar, left central, and left bicuspids (*fig. 23*). Vertical extension helped to maintain contour of face and to close perforation. *Dec., 1931:* patient seen again; no evidence of recurrence; wearing denture comfortably (*figs. 24* and *25*).

Case VI: Mr. H.—Patient seen, *Oct. 29, 1930*; operated two weeks before by Dr. C. Simmons for carcinoma of upper jaw. Examination: entire

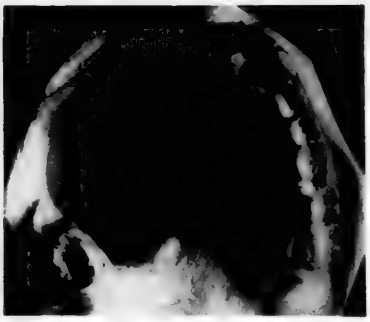


FIG. 22. MODEL SHOWING LARGE PERFORATION OF PALATE FOLLOWING RADIUM IMPLANTATION

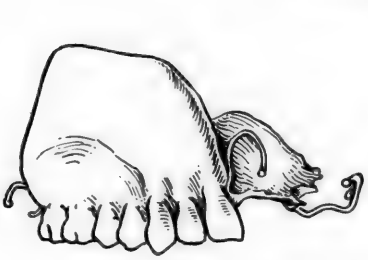


FIG. 23a

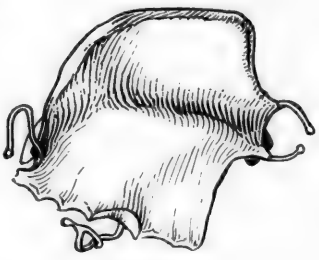


FIG. 23b

FIG. 23. DIAGRAM OF UPPER DENTURE

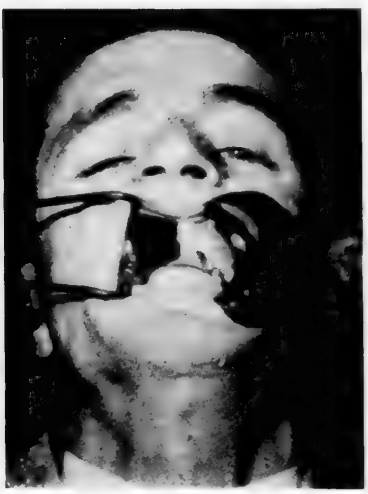


FIG. 24

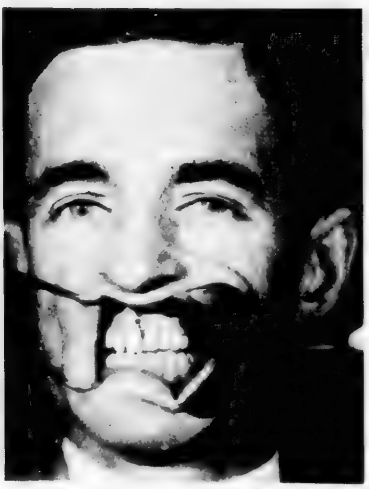


FIG. 25

FIGS. 24 AND 25. PATIENT WEARING DENTURE COMFORTABLY; NO EVIDENCE OF RECURRENCE

hard palate missing, leaving only rim of alveolar process (*fig. 26*). *Nov. 14:* temporary appliance made, to cover opening (*fig. 27*); consisted of vulcanite base plate, retained in position by two clasps over upper cuspids, anteriorly, and extension of vulcanite above border of soft palate toward

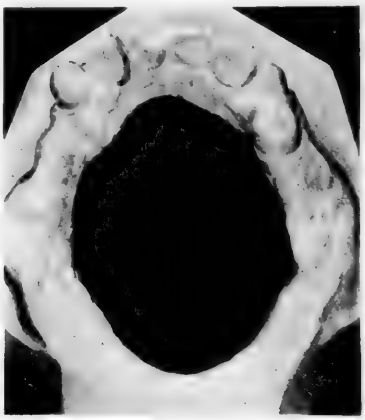


FIG. 26. MODEL SHOWING LARGE PERFORATION OF HARD PALATE FOLLOWING OPERATION

FIG. 27. VULCANITE BASE-PLATE; CONSTRUCTED SOON AFTER OPERATION

nasal pharynx, posteriorly (*fig. 27, a*); gave patient much comfort by closing perforation of nasal cavity. Soft tissues healed satisfactorily, while appliance was being worn. Later, artificial teeth added to denture to supply bicuspids and molars.[4]

[4] For further elaboration of this subject see Nichols: Prosthetic Dentistry, page 483.

Restoration of the mandible

In civilian life, most patients suffering from losses of mandibular structure are victims of neo-plastic diseases, although osteomyelitis and trauma play a noticeable part. Massive resection of the lower jaw is done for benign tumors such as adamantinoma, osteogenetic and giant-cell sarcoma, as well as malignancies. Unfortunately, if carcinoma of the jaw has advanced far enough to require massive resection, it is usually too far advanced for permanent cure. Thus restorative treatment for patients suffering from carcinoma is limited to only very favorable cases, but there are great restorative possibilities in deformities resulting from benign tumors.

Artificial restoration of the mandible has definite problems of its own. Unlike the maxilla, it depends on active movements for its function. The primary object is to preserve the functioning of the remaining part of the mandible, and to restore the face to its normal contour. The degree of functional power is dependent upon the size and anatomical position of the existing bone, and upon the presence or absence of teeth. Most restorations are from long-established deformities, and the general pictures of these cases may differ greatly. The remaining part of the mandible is usually distorted laterally and backward, disturbing the occlusion and the normal functioning of the jaw. The temporo-mandibular joint is completely filled with soft tissue, and the side of the face is more or less flat.

The first step is to correct the distortion of the mandible so that the remaining teeth will be in normal position. In favorable cases this may require only a certain amount of mechanical manipulation with orthodontic appliances; in others, complicated by the presence of cicatricial tissue, surgical interference becomes inevitable. Appliances to restore partial resection of the jaw can be justly called retention appliances. Their main function is not so much to correct facial contour as to facilitate the functioning of the remaining mandibular segment. Such a contrivance may be with or without teeth, and extends backward and upward toward the maxillary third molars, where a pseudo-temporo-mandibular joint is made in the form of either a groove, or ball-and-socket joint. Another method is the use of inclined planes. The fundamental purpose is to prevent the backward and inward swing of the remaining segment of the mandible.

Case VII: Mrs. McC.; age 25.—In 1924, operated by me for adamantinoma of mandible (*fig. 28*). Later, appliance made to control left half of mandible (*fig. 29*); consisted of ball-and-socket joint behind upper right third molar region (*fig. 29, a*), and series of bands attached to some of remaining lower teeth. Horizontal tube soldered to lingual aspect of lower incisors (*fig. 29, b*), and stout wire extended from tube to groove at lower third molar region (*fig. 29, c*). Appliance made hurriedly as temporary measure; two years later (*1926*) patient returned to clinic, still using appliance successfully (*fig. 30*); permanent appliance then made on same pattern by Dr. Henry J. Carney.

Immediate mandibular prosthesis has many points in its favor. Deformity and disability are avoided from the beginning. This method consists of introducing into the wound an artificial appliance, the approximate size and shape of the bone removed, extending from the articular eminence to the stump of the remaining mandible. The restoration stays in position until the wound has healed, but not infrequently skin grafting within the buccal cavity becomes necessary to avoid contractions. The final prosthesis is made with attachments to be fastened over the remaining teeth. This method was first advocated by Claude Martin, and has been elaborated and perfected by our European colleagues, including Schroeder,[5] Ernst,[6] Pichler[7] and others. Unfortunately this method has not so far been fully adopted in this country, mainly because of the reluctance of the general surgeon to follow this procedure.

Case VIII: Mrs. S.; age 34.—Patient first seen in *May, 1930;* chief complaint, tumor of lower jaw. Lower denervated second molar had been removed ten years before; soon afterward, lump appeared on side of jaw, gradually growing larger. Operated at local hospital, and tissue removed for biopsy. Second operation performed later gave temporary relief. One year ago swelling became more pronounced, and gradually grew larger until occlusion of teeth became affected. Local examination: reddish

[5] Schroeder: Die Anwendungsweise zahnärztlicher Prothetik im Bereiche des Gesichts mit besonderer Berücksichtigung des sofortigen Kieferersatzes nach Resektionen. *Korr. Bl. f. Zahnärzte*, 1901.

[6] Ernst: Prothesen nach Unterkiefer-resektion. Die Chirurgie, von Kirschner und Nordman, 1927, iv, p. 928.

[7] Pichler: Über Unterkieferresektionsprothesen. *Österr.-ung. Viert. f. Zahnheilk.*, 1914.

elevated mass, occupying greater part of lower left jaw, smooth and slightly lobulated; no palpable glands noticed in neck. X-ray picture: typical adamantinoma of lower jaw occupying almost entire half of ramus on left

FIG. 28

FIG. 30

FIG. 28. PHOTOGRAPH OF PATIENT BEFORE OPERATION
FIG. 30. APPLIANCE IN POSITION

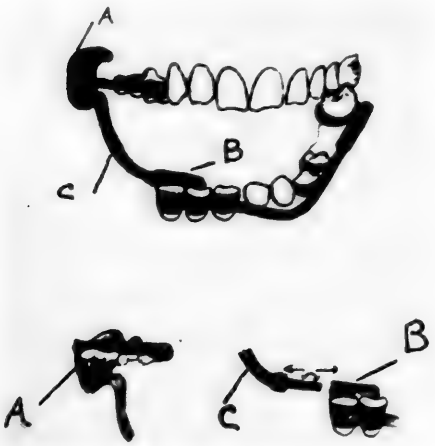


FIG. 29. RETENTION APPLIANCE TO HOLD REMAINING PART OF MANDIBLE IN PROPER POSITION

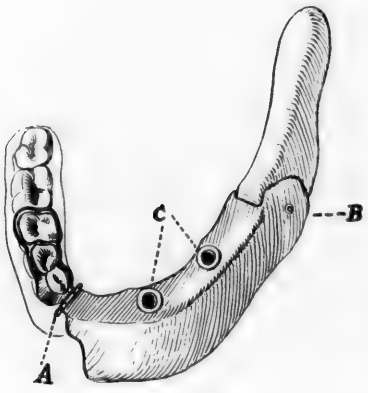


FIG. 31

FIG. 33

FIG. 31. PERMANENT PROSTHESIS FOLLOWING LATERAL RESECTION OF MANDIBLE

Auxiliary joints supplied at A and B, to allow appliance to adjust itself to movements of remaining part of mandible. Vertical tubes at C: for retention of denture carrying teeth.

FIG. 33. PATIENT WEARING PROSTHESIS

FIG. 32. X-RAY PICTURE OF APPLIANCE IN POSITION

side. Patient, under general anesthesia, operated by Dr. Herman and myself. Immediately after removal of tumor tissue, dental-compound mold shaped to approximate size of removal tissue placed in position; left in position for ten days. When removed, vulcanite temporary restoration inserted. When mouth wound healed, permanent appliance constructed and inserted (*figs. 31* and *32*). Patient seen, *Sep. 12, 1931;* no evidence of recurrence; wearing appliance satisfactorily (*fig. 33*).

During the World War it was conclusively demonstrated that for successful plastics of the lip, cheek, and chin, when destruction involved maxillary and mandibular bone, it was necessary for the prosthetic appliance to be constructed prior to the operation for repair of the part. This is also true in civilian life, with patients who have undergone operative treatment for removal of various tumors. In this instance we see the close relation of surgery and prosthesis; and on this basis, we may justly consider surgical prosthesis as one branch of restorative surgery.

II. SIMPLE PROSTHESIS

Arthur T. Rowe, D.D.S., Columbia University, New York City

I have been asked to speak briefly on the common or garden variety of dentistry. Simple dentistry may not be embodied in formula, nor may it be measured by rule. In the present day of growing realization of its very complexity, it seems almost a paradox that a group of scientific men should be interested in simple dentistry. The term does permit, however, of the stressing of fundamentals. It may be pertinent to point out that many of our present complexities have arisen through disregard or ignorance of these essentials. If dentistry could be practised ideally from a preventive standpoint, the dental office would become a service station to supply the oil and the slight carburetor adjustments that would keep the machine running smoothly. At present it is a garage where the horn can be jacked up and a new car put under it.

How the ideal practice may be arrived at is a question. Not so very many years ago people were warned not to eat tomatoes as they were the cause of cancer. Now we are advised to drink tomato juice to keep our teeth from decaying. Various methods of brushing the teeth have been advocated—up and down, crosswise, circular, etc.;

this or that tooth paste was the best; a clean tooth never decays; don't use dental floss; use dental floss; caries is caused by acid formed from decomposing food debris; the defence mechanism against decay is in the dental pulp; and so on, ad infinitum, ad nauseum. Dentists used to remove deposits from the necks of the teeth to prevent the attendant irritation to the gingival tissue. Now they write histories, make out charts, and draw up diet sheets to the point where the pencil sharpener is used more than the sterilizer. Trench mouth and "Vincent's" have been treated with Salvarsan to cure a condition that Cahn[8] has recently demonstrated to be the result of or closely associated with a fungus growth not affected by arsenic.

Medicine is just as inconsistent as dentistry. It is only recently that the medical profession has discovered that the human animal has a mouth. They have finally arrived there by way of the intestinal tract, and have specialties all the way up to the nose and throat. They are about to emerge into daylight through the mouth. It is time medical men began to realize that every tooth carrying a gold restoration is not necessarily pulpless; that a pulpless tooth may not be an infected one; and that an infected tooth does not always mean a primary focus. Ziskin has shown that the percentage of sick people with pulpless teeth is about the same as the percentage of well people with pulpless teeth, and that well people are also divided into about as many with pulpless teeth as without.[9] On the other hand, there is much evidence that individuals have suffered serious consequences from secondary infections with an infected tooth as the primary cause. The simplest way to solve the pulpless tooth problem would be to remove no more pulps. Carious and accidental exposures may in a fair percentage of cases be capped successfully. Where capping fails, extraction would be indicated. Attachments that require devitalization of abutments for their application have been pretty well discarded. But only two years ago a case presented where a patient had all pulpless teeth removed on advice of his physician, leaving two upper cuspids and two upper second molars as the only remaining healthy teeth. His dentist then devitalized all four of these teeth to use them as abutments. Balaam would still find a convenient conveyance.

[8] Cahn: *Dental Cosmos*, 1932 lxxiv, p. 21.
[9] Ziskin: *Journal of Dental Research*, 1931, xi, p. 285.

Dentists and physicians who practise on the basis of individual findings without the complete picture, so that the incidental factors may be properly evaluated, are empiricists, not scientists. Dentistry is not an entity, but it does occupy such a large place in the field of health service that it presents a sufficient problem to occupy individual attention. At present no one seems to be concentrating on the mouse trap—every one is trying to make the trip to the moon. A stricter limitation of the dental field, together with an intelligent and sympathetic coöperation between the various specialties of medicine, might go far toward solving the problem. Keen observation, coupled with a healthy curiosity as to the why and wherefore of what one is doing, is more apt to result in real education and study than a hodge-podge of scientific facts with a remote application to a problem to which one can bring no specific information for a solution.

In the field of restorative dentistry the problems are many. A misplaced contact point on a simple filling may lead to serious trouble. Closed bite or loss of normal facial dimensions, caused either by attrition or by shifting of the remaining teeth when some have been lost, may produce a series of untoward consequences. The change in directional stress applied to the teeth by the muscles in mastication may result in tortional and shearing stresses that cause periodontal inflammation and further loss of teeth. In the act of swallowing it is necessary to bring the teeth into contact. When the tooth length has become materially shortened, decidedly more effort is required to make this contact and the number of deglutitions is reduced. Mouth drainage is thereby reduced. The saliva lies for a longer time in the floor of the mouth and there is a greater precipitation of lime salts. Tartar deposits around the necks of the teeth are much heavier. Many patients have not only had the condition corrected by restoration of proper facial dimension, but the deposits that were present have been taken into solution and disappeared without any other treatment. Swallowing also produces a negative pressure that aids in the drainage of the sinuses. When swallowing is reduced, congestion is more apt to occur, producing an inflamed condition of the mucous membranes. When this inflamed condition spreads to the throat and eustachian tubes, enough congestion may be produced in the tubes to cause catarrhal deafness.

In any restoration of missing teeth it is necessary that physiologic function be restored as nearly as possible. Mechanical appliances of various kinds have been designed and employed to obtain fixation. Nearly all of them have much of good in a mechanical way, but are of value in application only insofar as they meet the biologic requirement of the case in hand. There is no ideal restoration per se, but there may be an ideal restoration for any particular case. No mechanical appliance is any better than the intelligence of the one who uses it.

Full-denture service is a still more complicated problem. Success is about 25 percent fit and 75 percent psychology. Mechanically all full-denture cases present difficulties, but some are harder than others. A case may be 100-percent perfect, mechanically, but of no value to the patient because he fails to coöperate and does nothing for himself. On the other hand patients present, balancing a denture on the end of the tongue, and declare it to be perfect in function. It might be well to say a word about "rubber sore-mouth." It is not! Metal dentures with rough tissue-contact surfaces that afford lodgement for microörganisms and debris produce the same condition. So will anemia. Scar tissue is still inelastic, and no method is known to me whereby a contracted post-operative lip can be stretched back into shape by the use of full dentures. Surgeons continue to ask that this be done, but a flap operation is the only solution.

While a great many prosthetic operations, and I include under that head all types of restorative dentistry, have been arrived at on a trial and error basis, a number of fundamental principles have been fairly well established by proceeding from effect to cause. An analysis of these fundamentals shows them to be well founded on biologic and mechanical facts. Black's concept of correct cavity preparation is not a difficult thing to learn so far as the mechanics are concerned, but an intelligent application requires a considerable biologic training. The theory of extension for prevention is sound insofar as inclusion of surface to relatively immune areas is concerned, no matter what theory one may hold as to the cause of caries.

Balanced occlusion is fundamental to full-denture prosthesis, not from a physiologic standpoint but from a purely mechanical one. Prosthetists have experimented with occlusion frames for years in an attempt to reproduce mandibular movement. They have argued the merits

of the various machines, drawn geometric designs, worked out check-bite systems, and mounting methods, until the construction of a denture has become a major undertaking. Recently one of the foremost contenders has come out with a mechanical tooth because his machine was so perfect it was impossible of use—a tacit admission that the problem was mechanical rather than anatomical. One is reminded of the argument of the group of medieval metaphysicians as to how many teeth a horse possessed. The controversy lasted for days. Each had his particular number and weighty argument to prove it correct. Finally one of them left the group for a few minutes and on his return announced the solution. Pressed for his proof, he said he had none but that he had gone out and counted them. Yet each articulator has behind it the thought of years of the man who designed it. He worked to produce an instrument that would interpret his desires of application. Could a dentist by using that man's articulator similarly express his desire? It isn't the articulator, it is the thought back of it—the principles, the facts of human articulation.

On the other hand, balanced occlusion in connection with bridge work is quite a different matter. Here it is as much biologic as mechanical, inasmuch as it is the application of a mechanical principle to a biologic problem in such a way as to distribute the load of mastication in a manner that will prevent the undue loading of any individual abutment tooth that would destroy the integrity of the whole. If one had a picture of the physiologic occlusion well established, might he not more readily detect the basic cause of tissue disturbances when that physiologic normal is disturbed? Might he not be expected to recognize change in facial dimension, the loss of muscle balance, and the many sequelae of bite closure?

There is such a multiplicity of factors in all that we do. Prosthesis is not just plugging cavities or filling spaces. If it is, we are treating symptoms not causes. Our treatment is not curative, it is reparative. But reparative treatment can and certainly should be preventive. The man who looks into a mouth and sees only a cavity in a tooth must have a strange conception of an ideal mouth. More has taken place there than the loss of a certain amount of tooth substance. How that loss is restored means the difference between return to an ideal or a continued retrogression. The ideal of the dental teacher is to

produce good dentists. The ideal of a good dentist is to promote and safeguard the health of those whom he serves. Wherein, it may not be illogical to point out, is the need for a concept of physiologic health that may be implanted in the mind of the undergraduate to be carried into mature years. This indelible picture of the healthy, normally functioning dental mechanism should go far toward the simplification of one's thought processes when dealing with abnormalities. Particularly is this so when the picture has a foundation of anatomical fact. The certain knowledge of the anatomical form and function of a part goes far toward dispelling vagaries of symptoms and cause. "There is a difference" in dentistry, and it is not one of methods or preliminary training, it is one of vision. G. V. Black had it, Tinker had it, Chayes had it; and so has every other man who has and is contributing to what dentistry really is and should be—the maintenance of the health and integrity of the oral cavity.

You are probably asking what the meaning of this rambling pessimistic paper may be. The answer is that with so many individuals in the various branches of health service possessing specific information on its several phases, the individual who tries to know it all will be as short of success as he who tries to be all things to all men. The most rudimentary thing that is done in dentistry may, if improperly done, be fraught with serious consequences. Let us concentrate on doing the thing we do in as nearly perfect a manner as may be, at the same time realizing our own limitations and availing ourselves of the help of other specialists who have concentrated on the thing they may be doing. I can no more conceive of an individual having enough specific knowledge of dental medicine to cover the entire field himself, than I can conceive of a man knowing enough to really be an atheist. Gentlemen, there is no simple dentistry.

III. CLEFT-PALATE PROSTHESIS AT THE COLUMBIA UNIVERSITY DENTAL SCHOOL CLINIC

H. A. Young, D.D.S., Columbia University, New York City

My part on this evening's program is to show the technic used at the Dental School Clinic for the prosthetic correction of cleft palate. The term cleft palate brings to a great many of the profession visions of some individual with deformed features. The features may be slightly

marred by a notched lip; or, if more extremely involved by a single or double harelip, the individual's appearance and facial expression are most displeasing to say the least. This confusion of the deformity of cleft palate with that of harelip seems to indicate the rarity of its occurrence in the average practice, especially in the smaller cities. Perhaps the confusion is due to the frequency of the occurrence of the two deformities together. The members of the profession more intimately acquainted with cleft palates visualize an individual whose palates are fissured to a greater or less extent, with a serious impediment of speech, and who experiences difficulty in the acts of mastication, insalivation, and deglutition.

The correction of cleft palate by prosthetic means is very old. Possibly the early attempts were simple appliances, conceived and executed by the patient himself, for the minor clefts more properly termed perforations—and perhaps for the single purpose of reducing the difficulty in mastication and deglutition. As time went on there was found a deeper appreciation in the individual of the ill effects of cleft palate, especially congenital partial and complete clefts of the soft palate, and an increasing desire to have the deformity corrected by some means. The medical and dental professions met this greater demand with an advancing knowledge of the deformity and of ways and means of correcting it. The correction of a congenital cleft of the soft palate is a problem entirely different from that of correcting a fissure of the hard palate only. We find it is not simply a bridging of the gap, and devoid of other considerations; rather it is a bridging of the gap in such a manner that the muscles functioning in the acts of deglutition, and speech control and projection, shall make contact with the edges of the appliances to form a valve seal, completely shutting off the oral cavity from the nasal and pharyngeal cavities at will, and without pain or discomfort during or following such muscle activity.

Within the last hundred years many technics and appliances have been presented, which may be grouped into two general classes: obturators and vela. Of the two classes perhaps vela have been employed the most. In the Clinic we employ obturators only, believing that the disadvantages of the vela are too many to balance their advantages. A velum is always in a more or less permanently septic

state, because of the marked rubber degeneration accompanied by the retention and decomposition of food and mucus. The velum soon becomes deformed, losing its vital elasticity with age, and therefore requires frequent renewals and consequent readjustment of the patient to a new appliance. Furthermore, we feel that because the action of the muscles of the soft palate in cleft-palate cases has been incorrectly interpreted, the basic principle of the appliance is faulty.

Slide 1 (*fig. 34*). I shall use this case to illustrate the various steps in the technic. You will note we have here a complete cleft of the soft palate with a partial cleft of the hard palate, involving approximately one-half of the hard palate. The first step in the construction of an appliance is to secure an accurate impression of the cleft and the parts necessarily involved to construct the remedial appliance, and to conserve the expenditure of time or labor of the operator, the patient, and the technician.

Technics of correction by prosthesis, as advocated in the past, required impressions of the parts of the soft palate at rest or with the muscles in a relaxed state, and the preferable impression material was plaster of Paris. The operation, therefore, has been limited to the more courageous and expert members of the profession largely because of two factors: *First*, the field of operation is at the most distal portion of the oral cavity, in fact at the very gateway to the air passages and alimentary canal, and therefore the danger of inhalation or ingestion of plaster fragments is increased, with possible serious results following such an unfortunate occurrence. *Second*, the accessibility of the anatomical parts necessitates the employment of a large mass of plaster, which flows into the nasal cavity, becomes locked, and usually is removed only with difficulty and with great discomfort. An additional discouraging influence is the probability that the impression step will have to be repeated, because a muscle activity of the patient registers an incorrect rest-position in the impression, and thus alters the shape and relative position of the pillars of the fauces. New and more easily controlled impression-materials are available today which, when used with the technic I am presenting, are practically devoid of these dangers and discomforts, and the operation is much easier to perform.

Slide 2 (*fig. 35*). We first take a dentocoll impression of the hard

palate area, the ridge, and teeth present, and, by using sufficient bulk of material (*slide 3, fig. 36*), obtain an indication of the lateral and antero-posterior extremes of the cleft except when the hard palate is completely fissured. We then cover the fissure of the hard palate with gauze to prevent the impression material from filling the nasal cavity. Such an impression would have all the detail of the above except the anterior extreme of the cleft. We use dentocoll impression-material because it has proved very accurate, is easily manipulated, and is more pleasing to the patient. Any specific materials mentioned are those we have found most useful in our hands, but we do not wish to convey the impression that they are the only ones suitable or available.

Slide 4 (*fig. 37*). From this impression we make a cast of artificial stone. Upon this cast we construct whatever retainers are deemed advisable, and model a base plate to be vulcanized and used to take the impression of the cleft of the soft palate. Associated with this deformity is a more or less constant irregular alignment, malformation and malocclusion of the teeth, making it very difficult to secure sufficiently opposed clasping areas to retain and stabilize an appliance in the mouth. To secure sufficient retention by clasping we must use some form of compound clasp. In the Clinic we have used at one time or another about every available form of clasp, and find that variations of the Jackson-crib clasp are more adaptable to the greater number of cases, and afford the maximum retention and stability.

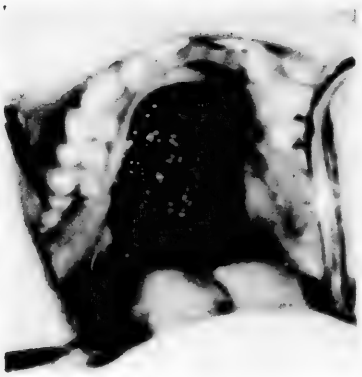
Slide 5 (*fig. 38*): the retention harness employed on this particular case. The one here illustrated was constructed of technic material instead of the proper spring gold-alloys, and is therefore not as neatly executed as the original. We usually employ a cast lingual-portion of the clasps, with 18- or 20-gage spring wire soldered on it, which forms the elastic arms.

Slide 6 (*fig. 39*): the tissue surface of the vulcanite base plate used to take the impression of the cleft of the soft palate. It is constructed by adapting one or two sheets of base-plate wax to the hard palate of the cast, allowing a tail-piece to project on a horizontal plane backward into the cleft from the point judged to be the normal ending of the hard palate to the back wall of the cast. *Slide 7* (*fig. 40*). A stud of wax is attached on the nasal surface about one-eighth of an inch from the end of the tail piece. This stud when replaced by vulcanite

is to aid in the retention of the bite wax during the various muscle manipulations necessary to get an impression of the cleft of the soft palate, for which we use the FitzGibbon method, utilizing a soft wax and muscle action to mold the impression into shape. This method is limited to the obturator type of remedial appliances. The vulcanite tail-piece is covered with sticky wax and a ball of Kelly's bite-wax attached, locking over the stud on the base plate.

Slide 8 (*fig. 41*). The wax-laden base-plate is now immersed in a pan of very warm water to insure softening of the wax throughout, then inserted in the mouth. The patient is asked to lower his head forward onto the chest as far as possible, and then to swallow two or three times. It is then removed, rewarmed, and reinserted in the mouth, and the patient asked to turn the head to one side, resting the chin on the shoulder, and then swallow two or three times again. Remove, rewarm, reinsert, and repeat the operation with the head turned and the chin resting on the opposite shoulder. The whole operation is repeated as often as is necessary, or until no further molding of the wax takes place. The excess of wax, in the molding, is forced forward into the nasal cavity or downward into the oral surface of the base plate. The excess that flows into the nasal cavity can serve no useful purpose, and its retention would only make the succeeding work most difficult. In fact it would be impossible to insert the finished obturator in the mouth when using mechanical retainers. When the impression is finished, we trim this excess portion off so that we have an indication of the posterior border of the hard palate, which will be sufficient to form our seal in this region. Likewise we have an excess impression of the naso-pharyngeal cavity. Upon examining the posterior part of the lateral sides of the impression we find a distinct elongated elevation, which is the mark of the orifice of the eustachian tube. The upper surface is trimmed off, on a slant downward and forward from the upper termination of this elevation, to the anterior notch formed by the posterior border of the hard palate. The excess on the oral surface of the base plate is of course trimmed off.

The appliance we construct is a modified Suersen-type obturator. Dr. Wilhelm Suersen advocated the use of the superior constrictor of the pharynx to make contact with the tail piece of the obturator and form the seal. This muscle in contracting draws the lateral walls



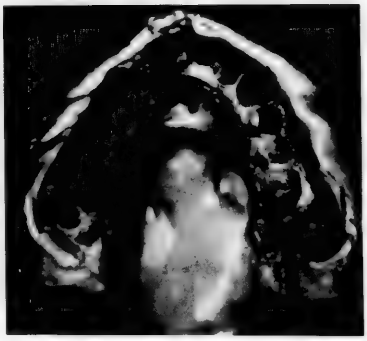


Fig. 34 Fig. 35

Fig. 34. Complete Cleft of Soft Palate, with Partial Cleft of Hard Palate, Involving Approximately One-half of Hard Palate

Compare with *fig. 52*

Fig. 35. Dentocoll Impression of Hard Palate Area, Ridge, and Teeth

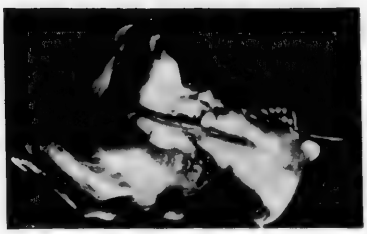


Fig. 36 Fig. 37

Fig. 36. Dentocoll Impression of Lateral, and Antero-Posterior Extremes of the Cleft Palate

Fig. 37. Cast of Artificial Stone: Made from Dentocoll Impression

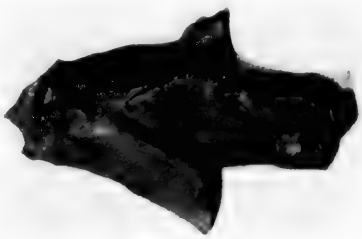


Fig. 38 Fig. 39

Fig. 38. Retention Harness

Fig. 39. Tissue Surface of Vulcanite Base-plate Used to Take Impression of Cleft of Soft Palate

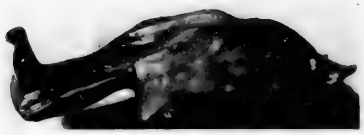


Fig. 40

Fig. 41

Fig. 40. Stud of Wax Attached on Nasal Surface (*fig. 39*) about One-eighth Inch from End of Tail Piece

Fig. 41. Wax-laden Base-plate Ready for Immersion in Warm Water before Insertion into Patient's Mouth for the Molding of the Wax

Fig. 42

Fig. 43

Fig. 42. Posterior Surface of Wax Impression

Fig. 43. Cast Trimmed for Flowing Second Part of Cast After Obtaining Impression of Cleft

Fig. 44

Fig. 45

Fig. 44. Complete Cast; Parts Fractured and Reassembled

Fig. 45. First Step in Construction of Model Obturator; Sheet of Base-plate Material Adapted to Cast; Only Half of Base-plate Trimmed

of the pharynx inward and bulges, forming a cushion on the posterior wall of the pharynx. The majority of obturators in the past have been constructed with a large mass of material extending into the naso-pharyngeal cavity. We believe this bulk of material obstructs the normal flow of air through the nasal cavity, and is reflected in mucus stagnation and low-grade degeneration of the nasal tissues. To lessen this obstruction, the Suersen obturator may be modified as much as may be required by removing most of the bulk, and by forming the nasal floor of the obturator to aid in the drainage of nasal mucus.

Slide 9 (*fig. 42*): the posterior surface of the wax impression. The shaded area in the lower center is the hollow formed by the Passavant cushion. We intend that our appliance shall come in contact with this cushion just below the height of bulge forming the posterior seal of our appliance.

Slide 10 (*fig. 43*). After the impression of the cleft of the soft palate is obtained and trimmed as we desire, we go back to the original cast and trim the cleft portion of it until the base plate carrying the impression of the cleft will seat in its correct position without interference. The base plate is then securely fastened to place with sticky wax and the remainder of the cast is formed in stone. The necessary surfaces of the original cast are painted with a separating medium before forming the second half. Since it will be necessary to split the second part of the cast, and also to remove it from contact with the original cast, we must have some means of retaining the original relation. *Slide 11* (*fig. 44*). To do this we paint the bottoms of the casts with a separating medium, and flow another base of stone upon it. Proper indentation should be made on the casts before pouring the base; this will aid materially in correctly positioning the parts later on. The parts of the stone cast are dissociated after hardening, and the two parts of the cast proper are split in the median line. This is done by first sawing most of the way through the casts, starting at the base surface; then, by using a knife blade or a thin instrument and exerting a prying force, the casts are split.

Slide 12 (*fig. 45*). The first step in the construction of our model obturator is to adapt a sheet of base-plate material to the cast. Half of it only has been trimmed in this slide. The trimming may be

done with vulcanite burs or files. The base plate is trimmed so that it covers the hard-palate area up to the gingival margin. The tail portion is trimmed to the edges of the cleft. It is desirable to let the base-plate material sag into the cleft portion about 4 mm. When the base plate is properly trimmed and cooled, we lay it aside and remove the posterior segments of the assembled cast. These segments along with base-plate wax are immersed in very warm water to soften the wax, and to heat and moisten the cast surface to prevent chilling and sticking of the wax when it is adapted. *Slide 13* (*fig. 46*). We adapt two separate layers to each half by means of a large pledget of cotton saturated with warm water; the parts and the wax are in the warm water during this operation. Two layers are used to allow for a certain amount of thinning of the wax. This wax is to form the lateral sides of our obturator, and is not lapped onto the upper or posterior walls of the cast.

Slide 14 (*fig. 47*). When the wax has been trimmed, tested for distortion, and thoroughly chilled, the small amount of necessary handling may be accomplished without distorting the model. The lower left illustrates the wax properly trimmed. The additional illustrations are steps in an experimental technic. *Slide 15* (*fig. 48*). One-half of the posterior part of the cast, having the base-plate wax in place, is assembled with the original cast and base. The trimmed base-plate material is placed in position; any interference existing between it and the base-plate wax is removed, and the two stuck together by tilting the assembled casts to one side and dropping some sticky wax along the line of their junction. The absence of half of the posterior segment affords ample access for free use of the wax spatula. After chilling the sticky wax, the base plate with the adherent base-plate wax is removed from the cast and, by running a hot spatula along the line of juncture on the outer side, the base-plate wax is completely and effectively sealed to the base-plate material. It is then tested for distortion and interference, and corrected if any exists. This part of the posterior segment is removed, and the same operation repeated with the second segment in position. With the tail piece modeled, we are ready to attach the mechanical retainers. The parts of the cast are assembled with the modeled obturator in place, and the metal harness is then heated to almost a red heat and

quickly seated in its intended position. By having the heat in the harness, and by using base-plate material, this may be done without fear of disturbing the work already completed. The sag in the palate is now filled in so that the vault will have a gentle harmonious curve.

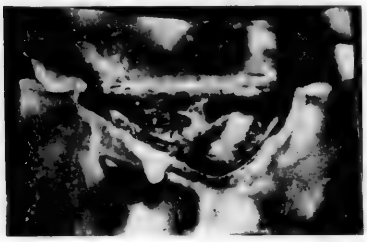


FIG. 46a

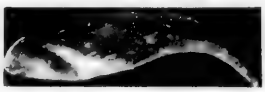


FIG. 46b

FIG. 46. TWO SEPARATE LAYERS ADAPTED TO EACH HALF BY MEANS OF LARGE PLEDGET OF COTTON SATURATED WITH WARM WATER

FIG. 47

FIG. 48

FIG. 47. LOWER LEFT ILLUSTRATES WAX PROPERLY TRIMMED
FIG. 48. HALF OF THE POSTERIOR PART OF CAST, WITH BASE-PLATE WAX IN PLACE, ASSEMBLED WITH ORIGINAL CAST AND BASE

After we have the retainer in place, the model obturator is removed from the cast completely, and at once a piece of base-plate material is attached with sticky wax across the free ends of the lateral sides to prevent distortion of the sides in the work that is to follow. To reduce the bulkiness of the obturator, to which I referred earlier, we have designed the obturator with a floor and two lateral walls of vulcanite

as thin as is reasonable for the case at hand. We construct the nasal surface of the floor in a definite manner because we believe there is bound to be a certain amount of mucus accumulation in the appliance.

FIG. 49

FIG. 50

FIG. 49. TINFOIL ADAPTED TO HALF OF TAIL PIECE OF MODEL OBTURATOR
FIG. 50. FURTHER PROGRESS IN ADAPTING TINFOIL TO MODEL OBTURATOR

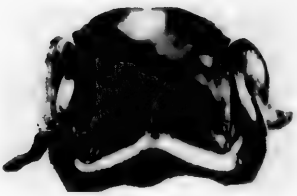


FIG. 51

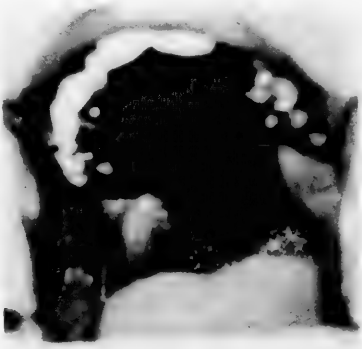


FIG. 52.

FIG. 51. POSTERIOR VIEW OF OBTURATOR; SHOWS TWO SIDES AND VERY POSTERIOR EDGE
FIG. 52. POSTERIOR VIEW OF APPLIANCE IN MOUTH
Compare with *fig. 31*

The nasal floor is constructed with an inverted V shape, the wings of the V forming a groove with the lateral sides. The apex of the V is in the median line, and runs straight back the full length of the tail piece. This groove aids in diverting the mucus to the posterior edge

of each side of the appliance, where it is automatically wiped off each time the muscles of the soft palate function. We make the inverted V-shape of the nasal floor by using a piece of base-plate wax of sufficient width to fill the space, warm the wax slightly to aid in positioning, and then with a spatula seal it along the lateral and posterior edges. This automatically forms a rough V-shape. The upper edges of the lateral walls are then rounded off. This edge has been cut a little thick and we allow this excess of wax, which melts off in thinning, to run down on the inside, because this portion will be finished after the case has been vulcanized.

Slide 16 (*fig. 49*). All the tissue surfaces are tinfoiled. This is done by first adapting tinfoil to only the tail piece of the model obturator. Usually a groove is formed by the posterior border of the hard palate and the anterior pillar of the fauces. To try to adapt tinfoil to the entire half of the model obturator at one time is impossible. Adapting tinfoil to this groove creates a pressure on the appliance, and distortion results. Also many wrinkles would be formed in the tinfoil, resulting in a rough and fissured case that would require a great deal of dressing down after removal from the vulcanizer, which is distinctly undesirable. To eliminate this, only one-half is tinfoiled at one time. The tinfoil is extended up higher than the lateral wall. *Slide 17* (*fig. 50*). After this is finished, the hard palate area is foiled, letting the edges run up into the retainers; they are then slit and folded down over the retainers to hold them in position when the model obturators are invested in the flask. The same thing is done along the edge of the hard palate, and of the upper and posterior edge of the tail piece. After this much is tinfoiled, the other side is foiled in the same manner and invested in the flask. Just before the last closing of the flask, that part of the mold in the top half of the flask is tinfoiled; this gives tinfoiled surfaces to vulcanize the appliance against, except in the interior of the tail piece. This portion does not come in contact with any tissue, so we can do whatever dressing may be necessary without fear of losing any valve seals or creating discomfort to the patient.

Slide 18 (*fig. 51*): posterior view of the obturator. It shows the two sides and the very posterior edge. The posterior edge has the inverted V-shape with more or less of a groove formed where the floor

meets the lateral walls. This directs the mucus to the posterior termination of the groove on each side. *Slide 19* (*fig. 52*): posterior view of the appliance in the mouth.

This technic is fairly simple, and many more practitioners could treat the deformity by following the technic as presented. Inasmuch as all patients suffering from this deformity must go through a course of instruction for speech training, I think this appliance answers the requirement equally as well if not better than the velum. Many claim that by employing the velum type of prosthesis an immediate and more pronounced improvement in speech is noted, and the permanent correction of speech is more rapid. Yet such men as Kingsley and Ottolengui claim that patients can speak more distinctly with an obturator than with a velum. Therefore it seems unnecessary to construct a velum when an obturator is as effective, practically, and gives clearer phonation. The construction of an obturator saves the operator the expenditure of the time, money, and skill necessary to construct the velum, and saves the patient the cost of the velum, and the time and discomfort incident to its construction. It also eliminates the wearing of an appliance of greater septic tendency, and of less stability in form.

IV. DISCUSSION

Irving W. Voorhees, M.D. (New York City): In doing cleft-palate operations, I have come to the conclusion that in the adult a prosthesis is probably the best method of closing an opening. I had an adult patient who was very anxious to do away with his prosthesis, and inveigled me into trying to close the opening. He had a very large congenital cleft, and only a little tissue to work with. I tried to use what there was, but it broke down. Then I noticed he had plenty of tissue on the nasal septum lying free above the cleft. I conceived the idea of making a flap from one side of the nasal septum, drawing it down into the opening, and stitching it to the edges of the cleft. It looked so fine when he went off the table that everybody was very much pleased, including myself. I continued to be pleased for about three days. In five days, the flap sloughed out. Then he went back to his prosthesis, and had a new one made. I think it is pretty well accepted that in children we like to do these surgical procedures as early as possible—at least before the child begins to talk, because he has to acquire a different method of articulation after an operation is done, assuming that he has already acquired so-called "cleft palate speech."

There is some question about the removal of tonsils and adenoids preceding a cleft-palate operation. Some surgeons prefer to leave the tonsils and adenoids; others take them out preceding operative work. I have tried both ways, but have not yet come to a conclusion whether it is better to do the one thing or the other. On theoretical grounds, I believe we ought to clear up all the infection we can in the nose and throat preceding any plastic operative procedure. If tonsils and adenoids are infected, as they very often are, they probably should come out before we undertake an operative attack.

A word as to the technic of the closure of the cleft, speaking now simply of the soft and hard palate. Most of the work I have seen has been done at the Manhattan Eye, Ear and Throat Hospital—some by Dr. Mackenty, some by Dr. Buckley. I have followed along with them, and perhaps have modified their technic a little bit. But we use the lead plates, one in the anterior incision and another in the posterior incision. Small pieces of lead are hammered out very thin, and are cut with a pair of scissors and introduced through the incisions. This lead strap is drawn together, but the ends are left long enough so they stick down into the mouth. When the tongue attempts to rise and threatens to do away with your sutures, it soon learns not to strike against these rough ends of lead. That is a protection against the tongue's breaking down the suture line. My other point is the use of two layers of sutures; that is to say, after the cleft is split into two layers, I place a nasal set of sutures; and then on the mouth side and oral surface, another set of sutures. These are interrupted. The last two cases I did with chromic gut. I have seen linen used, I have seen silk used, and other sutures that had to be removed. I thought I would try gut, "take a chance," and see whether we could "get by" with a suture that would remain long enough and then would be absorbed or destroyed by nature's efforts. Both of these cases closed completely. If any of you have tried this, I would like to know your opinion of it.

Irving J. Sperber, D.D.S. (New York City): There is nothing that I could add to Dr. Kazanjian's very thorough paper, except to emphasize the importance of immediate, although temporary, prosthetic restoration in cases that have had extensive oral operations. Not only does the temporary prosthetic appliance keep the remaining tissues in their proper position by preventing severe contraction and distortion, but the appliance is valuable to the patient from an esthetic viewpoint. By diminishing the severeness of the deformity, it eliminates prolonged hospitalization, thereby enabling the patient to return home within a day or two of the

operation. The temporary prosthetic appliance is also extremely valuable from a mental-hygiene standpoint, as it eliminates a great deal of psychic shock that the patient undoubtedly would experience if he went home with a severe facial disfigurement together with an inability to talk clearly or to masticate his food properly.

At the Manhattan State Hospital and at the Psychiatric Institute, of the Columbia University Medical Centre, our dental departments place particular importance upon the correction of facial and dental deformities. It has been observed that failure to correct facial and dental deformities after committment to the hospital may frustrate plans for mental rehabilitation, especially in attempts to socialize or stimulate individuals to seek contacts with real life. By "dento-facial deformities" we refer not only to those pathological conditions described by Dr. Kazanjian, but also to disfigurements resulting from protruded and rotated anterior teeth and other forms of malocclusion having a tendency to protrude the lips or to produce a condition of asymmetry.

Oral surgery and dental prosthesis are undoubtedly valuable adjuvants in our approach to patients with mental disorders. Patients having obvious dental deformities react to them as they would to any life situation that creates a serious feeling of inferiority with its well-known psychic sequelae. In the organic period, when emotional instability is so frequently prominent, we see the greatest incidence of dental deformities and cosmetic problems. It is obvious that in some cases the dental problems that I mentioned above are the immediate situations to which these patients react in characteristically exaggerated manner.

Ample clinical data definitely indicate that certain individuals become mentally unstable following an extensive oral operation where no attempt is made by the oral surgeon to give them immediate, if only temporary, appliances to tide over the healing period. These patients have acute problems of adjustment when, instead of being hospitalized, they are sent back to their homes or places of employment with severe dento-facial deformities. It has been our observation that certain of these patients, unless they receive sympathetic consideration from their families, drift away from their homes, going gradually into a state of depression. A great deal can be accomplished for them even at this stage. Given a proper restoration and sympathetic treatment, they can still readjust themselves. However, most patients at this time get into some difficulty by either attempting suicide, or by developing ideas of persecution, making it necessary for them to be committed to an institution for the safety of themselves and their families. We resort to flap and resection operations, or to pros-

thetic devices such as Dr. Kazanjian showed, to aid in a readjustment of the mental condition of these patients. We can thereby reduce their hospital residence to a comfortable degree.

An illustrative case may interest you. This patient [showing photograph not submitted for publication] was 73 years old when admitted to the hospital. He was a farmer and had a habit of smoking a pipe on the left side of his mouth while at work. He developed an epithelioma and was taken to the New York Skin and Cancer Hospital where a flap operation was performed to correct the deformity. He left the hospital before a second operation could be performed, but with the characteristic V-shaped deformity. He became a problem at home because of his mental state. He developed a marked depression. Added thereto was the difficulty he experienced in keeping saliva from drooling from the deformity onto his clothes. It was impossible for him to continue at profitable employment. His home life was far from ideal, in that his wife and daughters continually urged him to be cleaner in his oral habits, which it was impossible for him to do, owing to the constant oozing of the saliva through the V-shaped deformity. He attempted suicide. After commitment to Bellevue Hospital he was sent to the Manhattan State Hospital. On admission here, the patient felt that his condition was hopeless and that nothing could be done for him. The pyorrhetic and loose teeth were removed. A plastic operation was performed, closing the V-shaped deformity, and upper and lower dentures were constructed. He was advised to grow a beard. In a very short time the man's outlook on life seemed to change. We employed him usefully on the farm at the hospital, and through our social service department were able to secure employment for him. The man was discharged, and up to the present has been making a very nice adjustment. This picture [not submitted for publication] is a lateral view of the same man showing the improvement it was possible to make simply by removing some fat and tissue and closing the orifice of the mouth. Time does not permit more details on this phase of the subject.

In conclusion I wish to emphasize again the important rôle the oral surgeon and the prosthodontist can play in the field of mental prophylaxis through the construction of temporary but immediate appliances in cases that have had extensive oral operations.

Henry S. Dunning, D.D.S., M.D. (New York City): I wish to congratulate the Society on having three such splendid papers tonight. It was a great pleasure to hear my old friend, Dr. Kazanjian, talk. He is a fine combination—a surgeon and prosthodontist all in one. This idea brings up

the question of surgery versus prosthodontia and prosthesis in many of these cases. Dr. Rowe's paper was not quite as pessimistic as usual, but he was right when he said a man couldn't do it all. It takes a very clever man to do more than one thing well, but Dr. Kazanjian is one of the men who can.

During the war, the French general surgeons didn't think anything at all of dentistry. Men like Sibileau and others—famous plastic surgeons over there—early thought that they could bring their flaps together and not have any mechanical appliances or bone structures, or whatnot, to drape these tissues upon. Before the war was very old, they decided they had to use very extensively the prosthetic side of dentistry. It is a marvelous field—the combination of these two. It is very important indeed in the restoration of a human face. I know perfectly well that many of these people during the war developed psychosis, and wouldn't go home until they had their faces pretty well fixed up temporarily with prosthetic appliances.

I believe Dr. Kazanjian's presentation of this work is about as fine as we could possibly get. In the matter of closing the very large palatal defects, where there is possibly half a palate of normal tissue on one side, I should like to ask whether, in bringing over a delayed flap, he sometimes takes the flap off and throws it over and epithelializes the nasal surface of the flap, and then puts it back and in about ten days or so effects the closure. Has he ever epithelialized a palatal flap? It seems to me we tried it in one case, and I think it has great possibilities in closing large palatal defects.

The question of obturators versus surgery in adults is important. I believe the surgeon really ought to see these cases first. A patient of mine the other day, a postman 43 years old, had a very large, almost complete, cleft of the hard palate right up in the alveolar ridge and marvelous tissue which had never been touched. He had been wearing an obturator for fifteen or twenty years. At the hospital we closed it immediately; we got 100-percent closure. He said: "My God, if I had known this could have been done some years ago, I wouldn't have been a postman; I would have been a cop." He was "kicking himself" because he spent fifteen years of his life wearing a plate. I believe most of the adult palates can be closed, and think that most anatomical closures are very much better than prosthetic appliances, if they can be properly reconstructed or made serviceable palates with sufficient length.

Dr. Young's work is very fine indeed. I know a great deal about it because he has helped me out so much up at the Clinic. I think, with all due respect to Dr. Young, that his work is not very simple. I agree with

Dr. Rowe that there isn't anything simple about this work. It is very complicated and requires an expert in its field. In each field a man has to specialize more or less in that definite type of work, and it is very hard to do everything.

Victor H. Sears, D.D.S. (New York City): Dr. Kazanjian's excellent presentation stressed the necessity for immediate restoration in cases where restoration is to be made. I was hoping he would say more about that very important thing, because sometimes immediate restoration means making the restoration before doing the surgery. Dr. Dunning said that the surgeon should see the patient first. I agree with that, but think that often, even before the operation, the patient should see the prosthetist in order that the restoration may be made prior to the surgical part. I have in mind a case where, had that been done, a patient with a very small cleft in the palate would have been very materially aided. He kept stuffing something up there in order to be comfortable. In doing so, he widened the cleft so much before he got to the prosthetist that the opening had been very much enlarged. Immediate restoration should be made, it seems to me, at least should be contemplated along with the desirability of making all prosthetic preparations before the surgical part is attempted.

Alfred Michaelis, M.D. (New York City): I would like to ask about the process of the appliance that extends behind the soft palate. Some such projection appears to go into the pharynx. Wouldn't that cause a reaction and cough? We know that, in our throat and nose work, anything thrust into the pharynx tends to induce violent spasm.

Dr. Kazanjian (in conclusion): Dr. Dunning referred to the delayed flaps of the palatal tissues. I am aware of the fact that they have served quite extensively. Whenever the perforation is near the cheek, I always rely mainly on the cheek tissue to close the perforation. We have a lot of mucous membrane there we can spare very comfortably. When the perforation is in the center of the palate, naturally we are obliged to use hard, unyielding mucous membrane of the palate. Personally, I have not had very good results with delayed flaps.

Dr. Dunning, I believe, also emphasized the matter of closing cleft palates in adults. I think that is a very good procedure. There is an erroneous opinion that children's palates should be closed before they start talking, because after that it will be harder for them to get used to normal speech. But that isn't so. If the palate is elastic and well covered, the

patient will be able to talk very comfortably. If any healthy adult with cleft palate is willing to be operated on, we are willing to try it.

Dr. Sears spoke about immediate restoration. I agree with him that preparations should be made for cases that are indicated for the immediate restoration, that is to say, impressions should be taken, attachments or different parts made, and even sets of different appliances to meet emergencies of the operation should be assembled.

Answering Dr. Michaelis' question: Extensions into the soft palate do not cause gagging because we do not extend them far enough. It goes over for about 5 mm.—just enough to cover the surface. If that extension does not fit well the surrounding tissues, it will naturally irritate. We take special precautions to see that it fits very accurately. In that case it does not cause difficulty.

POST-ERUPTIVE CHANGES IN THE PERMANENT DENTITION OF DOGS FED RACHITOGENIC DIETS[1]

S. N. BLACKBERG, PH.D., AND J. D. BERKE, D.D.S.

Department of Pharmacology, College of Physicians and Surgeons, and Laboratory of Oral Histology, School of Dental and Oral Surgery, Columbia University, New York City

Dental abnormalities in experimental animals have been produced in many ways. Any severe alteration in calcium phosphorus metabolism may lead to a detectable change in the dental tissues. However, it was the purpose of our animal experimentation to simulate some of the conditions commonly encountered in man. Vitamins have assumed an increasingly significant rôle in nutrition; some seem essential for normal calcification of bone and teeth. Medical and dental clinicians have reported that in rickets there is often abnormality in tooth eruption and calcification. It was our purpose to investigate the changes in dentition that accompany the development of rickets in laboratory animals. In a previous communication[2] we described changes in the pulp, dentin, and enamel of young and adult rats and dogs on rachitogenic diets. In that paper destruction of odontoblasts, fibrosis, engorgement of the capillaries, and necrosis in the pulpal tissue were described. Poor calcification was noted in the dentin and enamel. In the present paper we describe a group of four dogs exhibiting progressive stages of rickets and concurrent tooth abnormalities.

Three puppies (345, 346, 347) were from the same litter; the fourth animal, 61, had no litter-mate in this experiment. After the permanent teeth had erupted, three of the four dogs were placed on rachitogenic diet 201 (modified Mellanby diet),[3] composed as follows

[1] Acknowledgment is hereby made of the financial assistance of the Commonwealth Fund in the pursuit of this study.

[2] Blackberg, S. N., and Berke, J. D.: Effects of ante-natal and post-natal deficiency of vitamin D on animal dentition, *Jour. Dent. Res.*, 1932, xii, p. 349; April.

[3] Mellanby, May: Diet and the teeth, Part I. His Majesty's Stationery Office, London, England; 1929, p. 142.

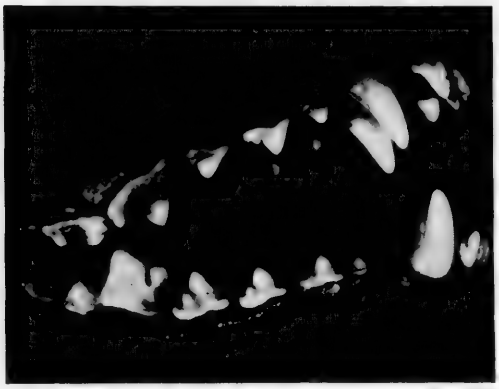


FIG. 1. NO PATHOLOGY OF JAWS VISIBLE AFTER EATING RACHITOGENIC DIET FOR 157 DAYS

Dark areas on molars and premolars are due to tartar. (Animal 346)

FIG. 2. PHOTOMICROGRAPH OF CROSS-SECTION OF CANINE

Note fibrosis and vascularity of pulp, absence of odontoblasts, and irregularity of dentin wall. (Animal 347).

a, Dentin. *b*, Pulp. *c*, Irregular predentin border. *d*, Engorged blood vessel. *e*, Fibrosed pulpal tissue.

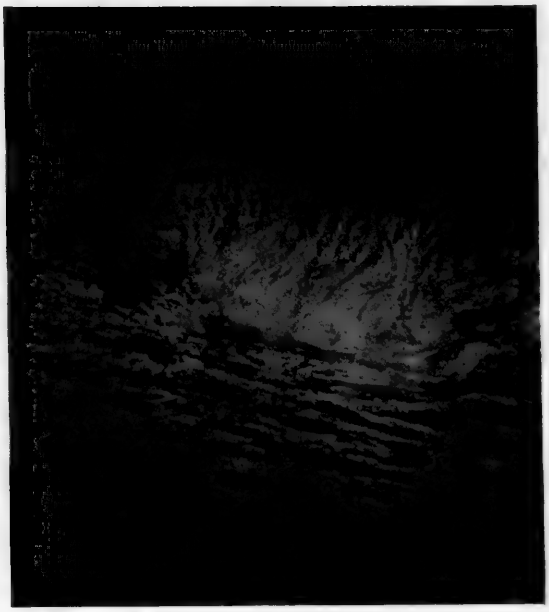


FIG. 3. PHOTOMICROGRAPH SHOWING FIBERS PASSING INTO DENTIN AT *d*

Note relationship between connective-tissue cells, *c*, of pulp and organic matrix.
a, Dentin. *b*, Pulp. *c*, Connective-tissue cells. *d*, Fibers constituting organic matrix of dentin.

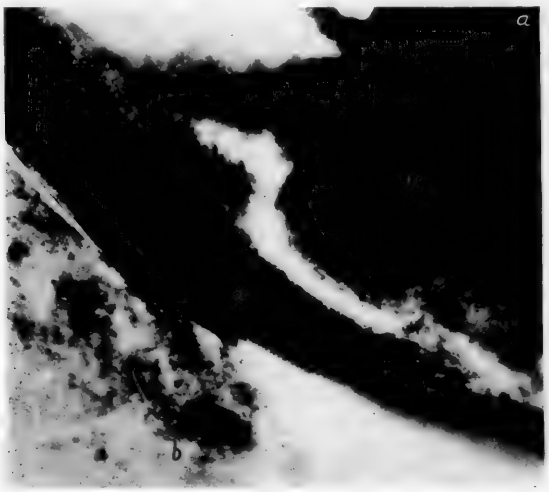


FIG. 4. MASSES OF TUBULAR PREDENTIN, *a*, SURROUNDED BY NECROTIC PULPAL TISSUE, *b*. (ANIMAL 347)

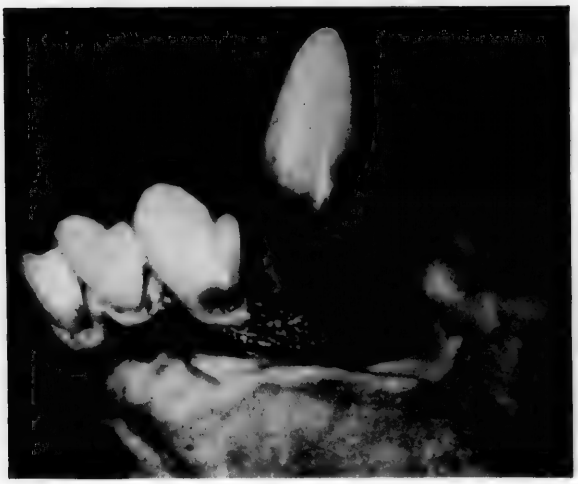
(parts): oatmeal, 360; skimmed milk, 50; Wesson oil, 15; linseed oil, 15; sodium chloride, 12; yeast, 15; meat, 50; tomato juice, 5 cc. per animal per day. The diet for the control group was the foregoing (201), plus Viosterol 250 D: 5 to 15 drops per animal per day, according to age and weight. Up to the time of the eruption of the permanent dentition, no gross dental pathologic changes had been noted. Animal 346 lived 157 days on the prescribed diet. Animal 347 was on the same diet for 201 days. Pup 345 was sacrificed twenty days later on the 221st day of feeding. Dog 61 had been on a bread and milk diet for 495 days when he was sacrificed. Ground and decalcified sections of the jaws were made. Our diagnoses of rickets were established by the clinical symptoms, x-ray findings, and determinations of calcium and phosphorus in the blood.

Animal 346 was the first to die after the experiment was initiated. Post-mortem examination revealed no unusual conditions. Histologic study of the tissues was not enlightening as to the cause of death. Macroscopically, the enamel and dentin of the teeth appeared normal. Tartar was observed on the molar and premolar teeth (*fig. 1*). The post-mortem changes in the pulp obscured any pathology that might have existed before death.

A picture of progressive rickets was presented by *pup 347*. The joints and costochondral junctions were enlarged. All of the usual pathognomonic symptoms of rickets were present. Gross examination of the teeth showed no abnormalities. Histologic study, however, revealed many changes. The odontoblasts were completely destroyed (*fig. 2*). In the pulp the reticular cells had lost their usual appearance, and took on the characteristics of fibroblasts; and the blood-vessels were engorged. The pulpal border of the predentin was very irregular, presenting a series of convexities and concavities (*fig. 2*). Only at the convexities were the connective-tissue cells of the pulp in intimate relationship with the predentin wall. The concave areas were rarefied, and showed numerous fibers passing between the dentin and the pulp. Some fibers passed quite a distance into the substance of the pulp (*fig. 3*); others ended near the border. The proximation of these fibers to the cells of the pulp was, in some areas, so close that the fibers seemed to be protoplasmic processes of the connective-tissue cells. Their direction was variable, passing into the dentin in a some-

what radiating manner. Most of these fibers seemed to be parallel to the dentinal tubules centrally, and diverged in the pulpal direction

FIG. 5. RADIOGRAPHS SHOWING EXTENT OF DECALCIFICATION OF SPECIMENS IN *figs. 6, 7, 8, and 9*
a, decalcified areas



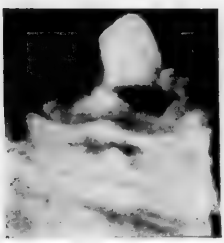


FIG. 6 FIG. 7

FIG. 6. PHOTOGRAPH OF LOWER CANINE, SHOWING LESION ON MESIAL SURFACE
Defect on distal cannot be seen. See x-ray in *fig. 5.* (Animal 345)

FIG. 7. BUCCAL ASPECT OF LESION IN PREMOLAR. (ANIMAL 345)

(*fig. 3*). The organic matrix of normal dentin has been described by Ebner, von Korff, Gebhardt, Ramon y Cajal, and by Mummery.[4]

[4] Mummery, J. Howard. Microscopic and gross anatomy of the teeth, 2nd edition, 1924; Oxford, England.

There was an unusual formation of predentin, in many of the specimens, appearing there as isolated masses completely surrounded by pulpal tissue. These islands presented the usual tubular structure of

FIG. 8. PHOTOMICROGRAPH OF CANINE, SHOWING REACTION IN DENTIN. SEE *figs. 5 and 6*

a, Lesions in enamel. *b*, Opaque dentinal tubules. *c*, Pulp

the tissue, and were isolated in the substance of the pulp (*fig. 4*). The possibility that these masses were fragments, broken off in the preparation of the sections, is precluded by the juxtaposition of the uncalcified dentin and fibrosed pulpal-tissue throughout the speci-

mens. The coronal portions of the pulps of most of these teeth were necrotic. Apically, the characteristic dilation of blood vessels, destruction of odontoblasts, and fibrosis can be seen (*fig. 2*). In one

FIG. 9. PHOTOMICROGRAPH OF PREMOLAR, SHOWING REACTION IN DENTIN

Note engorged vessels of pulp. See *figs. 5 and 7*
a, Lesion in enamel. *b*, Pulp. *c*, Opacity of dentinal tubules

specimen a pedunculated mass of dentin projected into the pulp canal. The enamel seemed to be normal in all the teeth of this animal.

Animal 345 was a litter-mate of 347 and 346. During life it presented a typical picture of rickets; autopsy revealed the usual rachitic changes. The initial dental sign was hypersensitivity of the upper and lower canines, followed by subsequent cupping of the enamel of the canines and premolars. These lesions gradually developed into well-defined depressions having a soft brownish base (*fig. 5*). A painful reaction was elicited on instrumentation at the base of the cavity. The lesions appeared on the mesial and distal surfaces of the cuspids (*fig. 6*), and on the bucco-occlusal surfaces of the premolars (*fig. 7*). Histologic examinations revealed a definite reaction in the dentin corresponding to that found in human caries, erosion, and abrasion (*figs. 8* and *9*). Bacterial plaques were found in these specimens (*fig. 10*), their presence eliminating the possibility that these lesions were associated with abrasion or erosion. Observed pulp changes coincide with those described above, except for the absence of masses of predentin in the tissue. The pathologic changes described for animal 347 were beautifully duplicated in these specimens. However, the dentin matrix (*fig. 11*) was even more evident than in animal 347. The histologic picture, and the radiolucency in the x-rays of the teeth, suggest an abstraction of calcium salts. *Fig. 12* is a radiograph of a tooth that showed no apparent lesion externally. The enamel was intact, and no perceptible discoloration was observed. The specimen was fixed in 10 percent formalin immediately after death. Histologic study disclosed destruction of odontoblasts, fibrosis, extreme engorgement of blood vessels, and marked degenerative changes in the reticular cells of the pulp (*fig. 11*). Radiographs revealed unusual radiolucency in the crown, although clinical tooth-destruction was not observed. The importance of these phenomena can be seen in *figs. 5, 6, 7, 8, 9*, where the extent of decalcification is far more penetrating than the lesions on the surfaces of the teeth.

Another type of dental lesion was seen in *dog 61*. The rachitis of this animal was as severe as in any of the others of this group. However, the extension of this experiment over a greater period of time may account for the marked changes to be described. The teeth of this animal displayed a lesion that has been commonly noted in rickets in man. The enamel of the crowns of the molars and premolars, and the tips of the incisors and canines, were severely altered

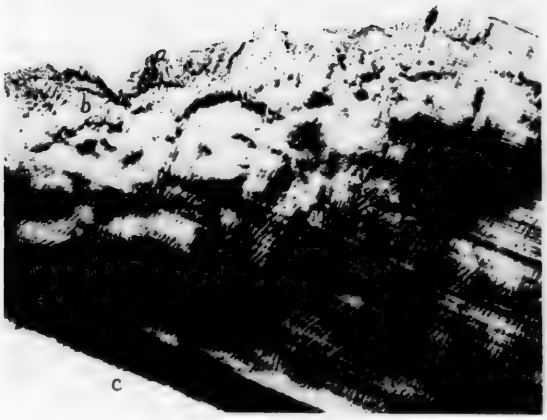


FIG. 10 FIG. 12

FIG. 10. PHOTOMICROGRAPH OF LESION IN ENAMEL, SHOWING BACTERIAL PLAQUE
a, Bacterial plaque. *b*, Etched enamel. *c*, Dentin border

FIG. 12. RADIOGRAPH OF CANINE, SHOWING RADIOLUCENCY IN CROWN WHERE NO LESION IN ENAMEL OBSERVED GROSSLY
a, Radiolucent areas. *b*, Radiopacity due to superimposition of palatal process

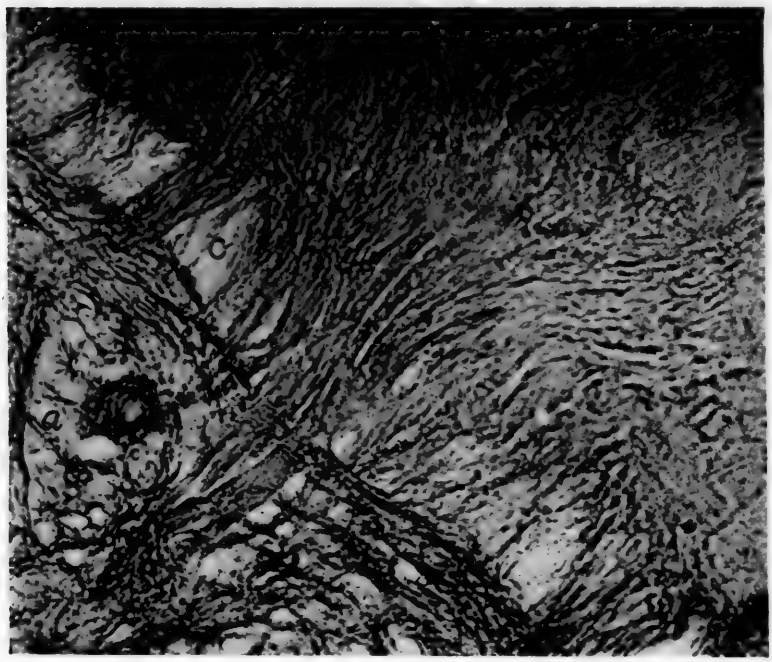


FIG 11. LONGITUDINAL SECTION OF CANINE PULP. BUNDLES OF FIBERS RADIATE FROM PULP, *a*, INTO DENTIN, *b*, AT *c*. (ANIMAL 345)

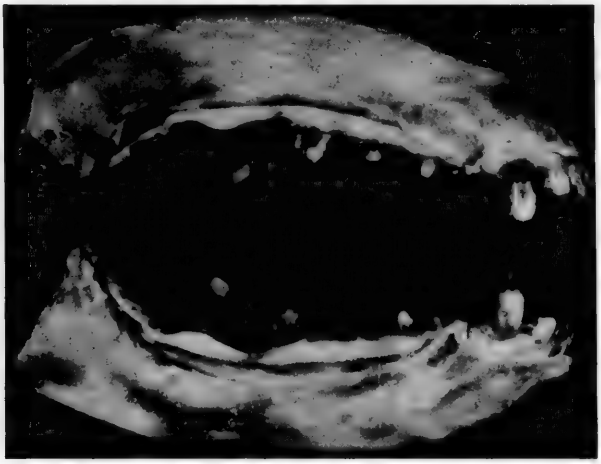


FIG. 13. PHOTOGRAPH OF MANDIBLE. ENAMEL MARKEDLY AFFECTED IN ANTERIORS AND POSTERIORS. (ANIMAL 61)

FIG. 14. PHOTOMICROGRAPH OF LOWER RIGHT CANINE, SHOWING EXTENT OF DESTRUCTION

Note engorged vessels of pulp. (Animal 61)
a, Enamel. *b*, Dentin. *c*, Lesions on tooth surface. *d*, Pulp

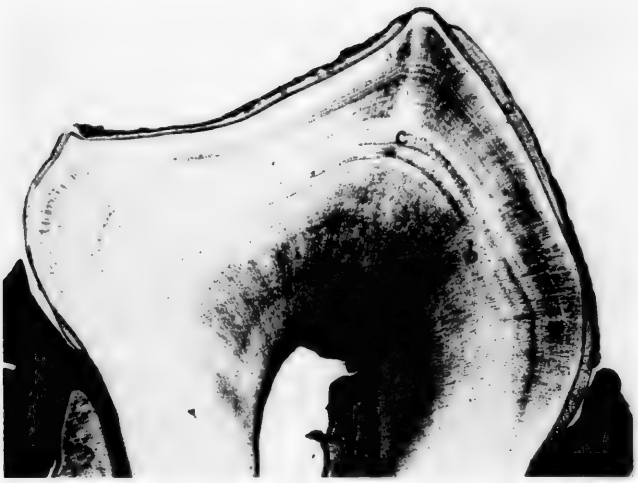


FIG. 15. PHOTOMICROGRAPH OF SECTION THROUGH MESIO-BUCCAL CUSP OF LOWER RIGHT FIRST MOLAR

Note extreme thinness of enamel cap
a, Enamel. *b*, Dentin. *c*, Interglobular spaces

FIG. 16. CORONAL PULP OF PREMOLAR

Note dilated capillary loops in close proximity to dentin wall. (Animal 61).
a, Dentin. *b*, Pulp. *c*, Capillary loops

(*fig. 13*). In some areas just a thin shell of enamel could be found; in others, the dentin was exposed (*fig. 14*). Clinically the areas of destruction appeared as large, brown, well-defined depressions. The

FIG. 17. PHOTOMICROGRAPH OF PULP, SHOWING EXTREME ENGORGEMENT AND RUPTURE OF CAPILLARIES

Note absence of odontoblasts.
a, Pulp. *b*, Dentin. *c*, Engorged vessels. *d*, Hemorrhagic area.

bases of the depressions were somewhat soft in some areas and hard elsewhere. Histologic examination revealed definite breaks in the enamel extending to the dentin. In some teeth the enamel was reduced to a thin layer surrounding the crowns (*fig. 15*). Bacterial

plaques were also present in the lesions described above. A remarkable change in the pulps of these teeth was observed. The dilated capillaries ended peripherally in a complicated mesh of capillary loops. The odontoblasts were destroyed, and replaced by a fibrous tissue. The capillary ramification was in proximity to the dentin wall (*fig. 16*). The capillaries more centrally located were extremely engorged, and in some areas ruptured (*fig. 17*). A few specimens show odontoblastic destruction in the coronal pulp only. Between the normal cells and those destroyed is a gradation of odontoblastic degeneration.

Summary

In the experiments described above, abnormalities, apparently resulting from the diets used, have been indicated. These rations were intended to upset the calcium and phosphorus metabolism. The lesions involved all the dental tissues. Extensive destruction of odontoblasts was seen in the pulps of the teeth of all four animals. Fibrosis, necrosis, extreme dilation of blood vessels, and abnormal predentin-formation, were found in the tooth pulps. The dentinal tubules beneath the lesions in the enamel were opaque. The pulpal border of the predentin was irregular, and the lack of calcium salts in those areas permitted the study of the organic matrix of the tissue. In view of the marked pathologic changes in the pulp, it seems probable that calcium has been removed from the radiolucent areas of the crowns, not by a process that begins at the free surface, but by a process that begins in the interior of the tooth itself. These changes have been produced in young animals after the eruption of the permanent teeth. The initial lesions seemed to be in the pulps, and changes in the dentin and enamel occurred only after prolonged dietary deficiencies. We are therefore of the belief that the duration of the experiments plays an important rôle in the development of changes in enamel. The presence of bacterial plaques, and opacity of the underlying dentinal tubules, are shown. Histologically, the enamel and dentin lesions described are suggestive of those in human caries.

We express our gratitude to Drs. C. C. Lieb and C. F. Bödecker for their suggestions in the preparation of this paper. We are indebted to Dr. E. Applebaum for the photomicrographs, and to Miss N. Hughes for help in sectioning the specimens.

A STUDY OF THE INCIDENCE OF DENTAL CARIES IN ONE THOUSAND CHINESE[1]

BERT G. ANDERSON, D.D.S.

Department of Surgery, School of Medicine, Yale University, New Haven, Conn.

In making routine dental examinations of patients applying for treatment at the Peiping Union Medical College, Peiping, China, the writer noted a comparative absence of decay in the teeth of the Chinese. This suggested a study of the incidence of dental caries among these people. There are no published reports to show that such an investigation among a large group of living Chinese has ever been made. This is a report of the findings in a study of dental caries in one thousand individuals, who, for the most part Chinese, lived in North China. There were also a few Mongols, and a scattering of natives of the Southern provinces residing in the North.

Examinations of small groups were conducted from time to time over a period of five years, beginning in 1924. Most of these groups were examined in Peiping and vicinity. In the groups were individuals from all social classes, including (1) school children, male and female, from the primary grades to and including Middle (High) School; (2) university students (chiefly males) residing in Peiping, but coming from most of China's eighteen provinces; (3) male factory workers; and (4) a heterogeneous group of individuals that came to the Dental Clinic of the Peiping Union Medical College for treatment. One group of 110, military-police students from various parts of the province of Shansi, were a rugged peasant-type ranging from 18 to 30 years of age. A small group of 25 young Mongols, male and female from 9 to 26 years of age, belonged ethnologically to the semi-nomadic

[1] The material for this paper was collected while the author was a member of the staff of the Peiping Union Medical College in Peiping China. A preliminary report was presented at a meeting of the New Haven Section of the International Association for Dental Research: *Journal of Dental Research*, 1931, xi, p. 235; Apr. The completed paper was presented at a meeting of the New Haven Section of the International Association for Dental Research, October 3, 1932.

Mongols living on the edge of the Gobi Desert about one hundred miles due north of Kalgan, a city on China's northwest frontier. The entire series is composed of 219 females and 781 males, 581 ranging from 5 to 15 years of age, inclusive; 307 from 16 to 25 years, inclusive; and 86 from 26 to 35 years, inclusive; and 26 being over 36 years of age. The ages are reckoned according to the Chinese method; according to our Western standard, each of these individuals would be approximately one year younger than is indicated by the Chinese figures.

The dentition of each individual was thoroughly examined by the writer with the help of a recording assistant. Each of the five surfaces of every tooth was carefully inspected, with the aid of a mouth mirror and an explorer. Teeth not seen in the mouth at the time of examination were recorded as missing, whether unerupted, extracted, or congenitally absent. No attempt was made to ascertain from an individual why a tooth was absent. However, it was evident that a very large portion of the missing teeth were simply unerupted. The youthfulness of so large a percentage of the whole group is sufficient to account for the discrepancy between the total number of individuals and the total number of teeth present.

Table 1 shows that 20,778 permanent teeth were present in the entire group. Of this number, 445 or 2.1 percent were carious. Of the 963 individuals having permanent teeth, 404 or 41.9 percent had dental caries of this dentition. The group having caries of the permanent teeth averaged 1.1 decayed teeth per individual. *Table 2* shows that a total of 5,173 deciduous teeth were examined. Of this number, 832 or 16.1 percent were carious; of the group having deciduous teeth, 51.0 percent exhibited caries of this dentition. The group having caries in the deciduous teeth averaged 3.6 decayed teeth per individual.

A consideration of these data, from the geographical standpoint, is also of interest. The examinations were made in three different localities. In Peiping and its immediate vicinity, 865 members of the entire group were examined; 376 or 43.4 percent of the number had dental caries. A second group of 110 members were examined in Tai Yuan Fu, in the province of Shansi; of this group 28 or 25.5 percent showed dental caries. A third group of 25 individuals, examined in Mongolia, were entirely free from dental caries. This third group was composed of young people who were attending a mission school in the

TABLE 1

Permanent teeth: Chinese

CONDITION		INDIVIDUAL TEETH																TOTAL	
Upper right	present	153	544	895	624	677	641	748	859	865	739	638	677	631	886	533	158	10,268	Upper left
	carious	15	12	30	7	3	1	0	2	2	0	2	3	5	28	17	13	140	
	symbol	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8		
Lower right	symbol	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8		Lower left
	carious	14	36	73	8	4	1	0	0	0	0	0	2	3	92	53	19	305	
	present	170	524	884	599	645	670	853	909	913	857	672	642	600	882	520	170	10,510	

Grand total: Teeth present, 20,778; teeth carious, 445

TABLE 2

Deciduous teeth: Chinese

CONDITION		INDIVIDUAL TEETH										TOTAL	
Upper right	present	375	310	345	185	118	115	185	338	311	370	2,652	Upper left
	carious	80	67	18	33	38	36	39	18	76	77	482	
	symbol	V	IV	III	II	I	I	II	III	IV	V		
Lower right	symbol	V	IV	III	II	I	I	II	III	IV	V		Lower left
	carious	81	80	11	1	1	1	1	5	90	79	350	
	present	395	341	307	133	83	78	135	319	339	391	2,521	

Grand total: Teeth present, 5,173; teeth carious, 832

district visited, and therefore could be examined systematically. The writer does not wish to give the impression that dental caries is unknown among the Mongols, since it was observed in the same region among patients attending a nearby clinic. However, dental caries does seem to be relatively rare among this race.

This study indicates that the incidence of dental caries decreases progressively as one travels from the port cities to the interior of China. This in turn suggests that the incidence of dental caries may be directly proportionate to the extent of modernization in manner of living.

MEDICO-DENTAL CASE RECORDS. XI. ENDOCARDITIS[1]

A Clinico-Pathological Study

BISSELL B. PALMER, D.D.S., F.A.C.D., and MALCOLM W. CARR, D.D.S.

New York City

CONTENTS

I. Introduction 713
II. Case C-7 (27): Endocarditis
General history and physical examination 714
Diagnosis and recommendations 715
Medical and surgical treatment 716
III. Follow-up observations 718
IV. Summary and discussion 719
V. Conclusions 720

I. INTRODUCTION

This is the third report, in our medico-dental case-record series, of a detailed clinical study of the relationship between cardio-vascular disease and oral sepsis. The first report presented a study of endocarditis; the ninth, presented a record of myocarditis in which an unusual sequence of events, due at least in part to emotional causes, led to fatal termination following removal of infected teeth. A comparative study of these three cases, particularly of the ninth report and the present one, is interesting. It would not be practical here to attempt a detailed comparison of these two cases. The patient whose history is described in the following pages had the same type of fear-and-anxiety complex as the patient described in the ninth report, although not to so pronounced a degree. Having recognized this condition in the present case, the special precautions of hospitalization with rest in bed and administration of digitalis several days before operation, the pre-operative use of morphine, the post-operative

[1] The previous reports were published in the *Journal of Dental Research*: (1) 1926, vi, p. 283; (2–4) 1927, vii, pp. 115, 275, 457; (5–6) 1928, viii, pp. 73, 579; (7) 1929, ix, p 89; (8–9) 1930, x, pp. 173, 675; (10) 1931, xi, p. 847.

administration of morphine and sedatives, operation in several stages (a simple procedure with minimum emotional shock at the first operation), and all operations performed in bed, were deemed advisable. In addition to the usual clinical and laboratory observations, made routinely for patients who are included in these focal-infection studies, a series of observations of blood pressure is reported in this case. In another series of cases now being compiled by one of the authors, and to be reported later, routine blood-pressure observations were made in a number of cases in an attempt to ascertain the relation of emotion, and particularly the major emotions of fear and anxiety, to blood pressure. This subject was also discussed briefly in the ninth report.

II. CASE C-7 (27): ENDOCARDITIS

General history and physical examination

Patient, male, 32 years of age; clerk; first seen in consultation, *Oct. 1925*.

Chief complaint. Numerous pulpless and infected teeth; also conscious of cardiac condition described by patient as "thumping of heart." Had been advised to have infected teeth removed because considered possible etiological or aggravating factors in existing heart condition. Patient apprehensive that operation for removal of infected teeth would involve considerable risk in present state of health, which he regarded as serious.

Family history. Father and mother both living and well; three brothers and one sister living and well. No family history of heart disease.

Past medical history. No history of rheumatic fever, chorea, muscle or joint pains, tonsilitis, or pneumonia. Never had diphtheria, typhoid fever, nephritis, or hyperthyroidism; had measles, and mild attack of scarlet fever in childhood. Participated in sports during youth; did considerable running; also played basketball; engaged in no sports after 17.

History of present illness. First became aware of heart condition in 1917, when so advised by private physician. In 1918, after ingestion of aspirin gr. x, awoke from sleep with profuse perspiration, palpitation, dyspneoa, and rapid pulse. Confined to bed for ten days with "*nervousness* and palpitation." Said he was relieved by osteopathy. Similar nervous attacks every three months until marriage two and one-half years ago. Two years ago dentist advised that several infected teeth should be removed. One tooth removed at that time; next day patient had irregular heart action, palpitation and dyspnoea. Under care of physician for one week; heart action then became regular. Unwilling to have remaining

infected teeth removed because of apprehension. Past year, patient felt nervous, rested poorly, fatigued easily; conscious of palpitation, dyspnoea, and irregular pulse action; "dizzy spells," with mental confusion and "dazed feelings," as if blood pressure too high. Owing to fatigue, unable to pursue usual active duties in business.

Habits. Slept from six to eight hours nightly, and rested moderately well, until one year ago. Appetite uniformly good. Lost no weight. Not addicted to use of alcohol, drugs or tobacco. Drank moderate quantities of tea and coffee.

Physical examination. Well-developed and nourished, pale but not cyanotic, nervous individual. *Eyes:* both reacted normally to light and accommodation. *Nose:* no obstruction, septum not deviated, and turbinates normal. *Mouth: clinical examination*—moderate deposition of salivary calculus, simple gingivitis, many filled teeth, several crowned teeth; *roentgenographic examination*—right maxillary first molar pulpless, crowned, incompletely filled root canals, chronic rarefying osteitis with granuloma and chronic periodontoclasia. Right maxillary second premolar pulpless, incompletely filled root canal, chronic rarefying osteitis. Right maxillary lateral incisor pulpless, incompletely filled root canal, cyst at apex. Right maxillary central incisor, left maxillary central incisor, lateral incisor, and canine, pulpless, incompletely filled root canals, with various degrees of rarefying osteitis at apices. Left maxillary first premolar had been removed. All other teeth and surrounding bony structure appeared normal. *Pharynx:* tonsils moderately enlarged, but did not appear infected. *Chest:* left thorax more prominent than right; expansion good and equal. Chest resonant throughout, breathing vesicular, no râles. *Heart:* rate 84, regular and good quality. Apex maximum impulse 8 cm., from median line in fifth space; lower and outer point 9.5 cm., from median line in sixth space. Diffuse pulsation, and whole chest pulsated with systolic pulsation, transmitted to muscles of neck. Systolic nodding of head. $A_2 > P_2$. First sound at apex roughened (++). Long diastolic murmur heard at base, short rough systolic murmur heard at apex, and short rubbing systolic murmur heard at base. No tenderness in precardium. Blood pressure: systolic, 180 mm.; diastolic, 40 mm. No sign of heart failure. *Liver:* not enlarged and not tender. *Spleen:* not palpable. *Extremities:* no pain, swelling or redness of joints, no history of arthritis, no edema.

Diagnosis and recommendations

(a) Endocarditis. *Etiological:* rheumatic and (scarlet fever?). *Anatomical:* aortic insufficiency and cardiac enlargement (++), especially left

ventricle (mitral insufficiency not present, and aortic stenosis probably not present—A_2 clear). *Physiological:* normal mechanism. Palpitation due to emotional causes. (b) Oral sepsis: seven pulpless teeth all showing chronic rarefying osteitis, one showing cystic degeneration.

Recommended removal of pulpless and infected teeth, in several stages; also, to overcome emotional strain and apprehension, that patient be admitted to hospital for rest in bed several days before first operation for removal of teeth, patient to receive digitalis. Admitted to hospital[2] *Nov. 21, 1925.*

Medical and surgical treatment

Required to rest quietly in bed for two days before operation. Received regularly digitalis three times a day; efforts made to overcome emotional stress.

First operation; Nov 23: temperature, 98.2; *pulse rate,* 73; *respiration,* 20. *Urinalysis:* specific gravity, 1.022; reaction, alkaline; albumin, sugar, casts, absent; leukocytes, epithelial cells, occasional. *Hematology:* hemoglobin, 110 percent; red blood-cells, 5,410,000; white blood-cells, 7,600; polymorphonuclear leukocytes, 52 percent; lymphocytes, 36 percent; transitionals, 4 percent; eosinophiles, 8 percent. *Blood chemistry* (mg. per 100 cc.): urea nitrogen, 13.9; creatinin, 0.8; uric acid, 2.1, sugar, 106; CO_2-combining power, 52 percent; complement-fixation test, negative. Given hypodermatically, half-hour before operation, morphine sulphate, gr. 1/6. Operation performed with patient in bed in semi-supine posture. Right maxillary second premolar removed with local anesthesia (procain 1 percent with suprarenin 1/40,000); 2.5 cc. injected slowly by infiltration technique. No unfavorable reaction to injection. *Pulse and blood-pressure observation:* pre-operative—pulse rate, 73; systolic blood-pressure, 140 mm.; diastolic, 58 mm. Three minutes after injection—pulse, 72; systolic blood-pressure, 150 mm.; diastolic, 38 mm. Fifteen minutes after injection (five minutes after operation)—pulse rate, 77; systolic blood-pressure, 145 mm.; diastolic, 40 mm. Right maxillary second premolar pulpless, incompletely filled root canal with chronic proliferative pericementitis and chronic rarefying osteitis. Culture: streptococcus viridans. Wound dressed with iodoform gauze. At 10 p.m. given post-operatively sodium bromide, 20 grains, well diluted in water. No exacerbation following operation. Experienced little emotional strain and only slight apprehension. Dressing changed next day; pulse rate then 64; blood pressure, systolic, 138 mm.; diastolic, 20 mm.

[2] The Fifth Avenue Hospital, New York City.

Second operation; Nov. 25: temperature, 98; *pulse rate,* 60; *respiration,* 20. *Hematology:* hemoglobin, 105 percent; red blood-cells, 4,910,000; white blood-cells, 10,200; polymorphonuclear leukocytes, 51 percent; lymphocytes, 39 percent; transitionals, 8 percent; eosinophiles, 2 percent. Given hypodermatically, half-hour before operation, morphine sulphate, gr. 1/6. Operation performed with patient in bed in a semi-supine posture. Right maxillary first molar removed with local anesthesia (procain 1 percent, with suprarenin 1–40,000); 2.5 cc. injected slowly by infiltration technique. No unfavorable reaction to injection. *Pulse and blood-pressure observation:* pre-operative—pulse rate, 60; systolic blood-pressure, 138 mm.; diastolic, 30 mm. Three minutes after injection—pulse rate, 64, although full and bounding; systolic blood-pressure, 152 mm.; diastolic, 28 mm. Twenty-five minutes after injection (five minutes after operation)—pulse rate, 64 (full and bounding); systolic blood-pressure, 138 mm.; diastolic, 30 mm. Right maxillary first molar pulpless, with incompletely filled root canal; chronic rarefying osteitis and granuloma formation at apex. Culture: streptococcus viridans. Wound dressed with iodoform gauze. At 9 p.m. given post-operatively morphine sulphate, gr. 1/6. No exacerbation following operation. Experienced no apprehension or anxiety, either before or after operation.

Third operation; Nov. 28: temperature, 98,4; *pulse rate,* 68; *respiration,* 20. *Hematology:* hemoglobin, 107 percent; red blood-cells, 5,350,000; white blood-cells, 15,000; polymorphonuclear leukocytes, 57 percent; lymphocytes, 40 percent; transitionals, 1 percent; eosinophiles, 2 percent. Given hypodermatically, half hour before operation, morphine sulphate, gr. 1/6. Operation performed with patient in bed in semi-supine posture. Left maxillary lateral incisor and canine removed with local anesthesia (procain 1 percent with suprarenin 1–40,000); 2.5 cc. injected slowly by infiltration technique. No unfavorable reaction to injection. *Pulse and blood-pressure observation:* pre-operative—pulse rate, 68; systolic blood-pressure, 130 mm.; diastolic, 43 mm. Five minutes after injection—pulse rate, 88, irregular; systolic blood-pressure, 143 mm.; diastolic, 43 mm. Twenty minutes after injection (ten minutes after operation)—pulse rate, 66, regular; systolic blood-pressure, 140 mm.; diastolic, 38 mm. Left maxillary lateral incisor pulpless; incompletely filled root canal and chronic rarefying osteitis, granuloma formation at apex. Culture: "no growth." Root canal of canine incompletely filled, and root showed chronic proliferative pericementitis; not cultured. Wounds dressed with iodoform gauze and at 9 p.m. patient given, post-operatively, morphine sulphate, gr. 1/6, patient apparently well accustomed to procedure; experienced no apprehension or anxiety; no exacerbation followed operation.

Fourth operation; Dec. 2: temperature, 98.2; *pulse rate*, 72; *respiration*, 20. *Hematology:* hemoglobin, 94 percent; red blood-cells, 4,870,000; white blood-cells, 11,200; polymorphonuclear leukocytes, 63 percent; lymphocytes, 22 percent; transitionals, 8 percent; eosinophiles, 5 percent; basophiles, 2 percent. Given hypodermatically, half hour before operation, morphine sulphate, gr. 1/6. Operation performed with patient in bed in semi-supine posture. Left maxillary central incisor, and right maxillary central and lateral incisors, removed with local anesthesia (procain 1 percent with suprarenin 1–40,000); 3.5 cc. injected slowly; anesthesia produced by infiltration technique on left side, by regional block of right infraorbital nerve on right side. No unfavorable reaction to injection. *Pulse and blood-pressure observation:* pre-operative—pulse rate, 72; systolic blood-pressure, 135 mm.; diastolic, 30 mm. Five minutes after injection—pulse rate, 108, increase in force and volume, regular in rhythm; systolic blood-pressure, 170 mm.; diastolic, 35 mm. Forty minutes after injection (at completion of operation)—pulse rate, 96; systolic blood-pressure, 135 mm.; diastolic, 60 mm. Left maxillary central incisor showed chronic proliferative pericementitis. Right maxillary central incisor pulpless, with incompletely filled root canal showing chronic rarefying osteitis at apex. Right lateral incisor pulpless; incompletely filled root canal and chronic rarefying osteitis, with cystic degeneration at apex. Wounds dressed with iodoform gauze. Patient seemed slightly more apprehensive, but no exacerbation ensued. Remained in hospital two days following operation; no untoward sequelae; discharged *Dec. 4.*

III. FOLLOW-UP OBSERVATIONS

Dec. 4, 1925. Hematology: hemoglobin, 107 percent; red blood-cells, 5,360,000; white blood-cells, 9,000; polymorphonuclear leukocytes, 64 percent; lymphocytes, 30 percent; transitionals, 4 percent; eosinophiles, 2 percent. *Blood chemistry* (mg. per 100 cc.): urea nitrogen, 10.9; creatinin, 0.8; uric acid, 2.0; sugar, 107; CO_2-combining power, 53 per cent. Seven infected teeth removed without untoward sequelae since entry to hospital, Nov. 23 (two weeks), and discharge (December 4).

June 30, 1926 (*six months*). Heart: elicited certain definite improvement over condition when first seen, principally in narrower pulse-pressure. In October, systolic blood-pressure, 180 mm.; diastolic, 40 mm.; at first follow-up (June) systolic blood pressure, 150 mm.; diastolic, 60 mm. Diffuse pulsation disappeared. Aortic insufficiency and moderate cardiac enlargement persist, but no indication of advancement of cardiac disease. Subjectively patient considerably improved; general health better; no symptoms

of dizziness or mental confusion; fatigue, malaise, and lassitude disappeared; able to carry on usual activities without fatigue; rested better, and subjective symptoms of palpitation and dyspnoea disappeared.

December 14, 1926 (one year). Since removal of infected teeth, general improvement persists. Usual active schedule followed without emotional or physical strain. No old symptoms. Blood pressure: systolic, 150 mm.; diastolic, 60 mm. Anatomically no change in condition of heart. Aortic insufficiency chief lesion. Stenosis doubtful. No further increase in heart size. Lesions apparently not progressive.

IV. SUMMARY AND DISCUSSION

A man thirty-two years of age had developed, within a period of one year, subjective symptoms of cardio-vascular disease and general toxemia. There were symptoms of malaise and lassitude, fatigue developed easily, and he rested poorly. He had symptoms of palpitation, dyspnoea, and irregular pulse. At times he felt mentally confused and occasionally had dizzy spells. The family history and past medical history were negative. Physical examination revealed aortic insufficiency, moderate cardiac enlargement (principally the left ventrical), and hypertension ($\frac{180}{40}$). There were no signs of decompensation or heart failure. Physical examination otherwise was negative. Search for focal infection revealed oral sepsis (seven pulpless and infected teeth). No other foci of infection were discovered. The infected teeth were considered an aggravating factor in the patient's general condition, and their removal was recommended.

Because of the existence of a rather pronounced fear-and-anxiety complex, special precautionary measures were used to relieve all emotional strain. The patient was accordingly admitted to the hospital several days before operation, for rest *in bed*. During this time the patient became familiar with hospital routine and less apprehensive. Digitalis was administered. The infected teeth were removed in four stages; morphine as a pre-operative medication was given in order to reduce emotional stress, and either morphine or sedatives were given post-operatively. Operations for the removal of the teeth were all performed in the patient's room, with the patient in bed in a semi-supine posture. Procain was used with a minimum admixture of suprarenin. There were no unfavorable reactions to

the use of procain (see summary of blood-pressure observations, *table 1*), the general reaction to the removal of the teeth was favorable (see summary of hematology, *table 2*), and no exacerbations followed any of these operations. The follow-up observation showed an improvement in the cardio-vascular system, principally in a reduction of the hypertension, a narrowing of pulse pressure, and an indication

TABLE 1

A comparative study of blood-pressure reactions during the course of each of the four operations

Systolic and diastolic pressures, expressed in millimeters of mercury, are recorded for each observation

DATE	BEFORE INJECTION	THREE MINUTES AFTER INJECTION	FIFTEEN MINUTES AFTER INJECTION
November 23: first operation	140	150	145
	58	38	40
November 25: second operation	138	152	138
	30	28	30
November 28: third operation	130	143	140
	43	43	38
December 2: fourth operation	135	170	135
	30	35	60

TABLE 2

A comparative study of the reactions of the cellular elements in the blood to the several operations for removal of infected teeth, as indicated by differential blood counts

DATE	HGB.	R.B.C.	W.B.C.	POLYM.	LYMPH.	TRANS.	EOSIN.	BASO.
November 23	110	5,410,000	7,600	52	36	4	8	
November 25	105	4,910,000	10,200	51	39	8	2	
November 28	107	5,350,000	15,000	57	40	1	2	
December 2	94	4,870,000	11,200	63	22	8	5	2
December 12	107	5,360,000	9,000	64	30	4	2	

that original lesions were not progressive. There was complete relief of all subjective symptoms of toxemia and cardio-vascular disease.

V. CONCLUSIONS

(1) Oral sepsis may constitute an active focus of infection and, as such, contribute to or aggravate cardio-vascular disease, particularly when of the rheumatic type.

(2) When symptoms of cardio-vascular disease (rheumatic carditis) exist concomitantly with symptoms of toxemia, it is highly desirable to eliminate every possible dental focus of infection.

(3) Precaution is recommended in the surgical treatment, for all cardiovascular cases, particularly those eliciting the fear-anxiety syndrome. Every care should be exercised to reduce to a minimum all emotional stress.

667 Madison Avenue

EXPERIMENTS ON THE DIETARY CONTROL OF DENTAL CARIES IN CHILDREN[1]

EWING C. McBEATH, D.D.S., B.S., M.D.

School of Dental and Oral Surgery, Columbia University, New York City

Probably the commonest of diseases which afflict civilized man is dental caries; and its ravages are more widespread in each succeeding generation. Statistics show that today probably 95 percent of school children suffer in varying degrees from tooth decay. It has been recognized of late, moreover, that dental caries, while a disease in itself, is also very often a symptom of serious or potentially serious systemic conditions. The continual increase in its prevalence constitutes a menace that grows more threatening, almost from year to year. The ultimate aim in the study of this disease is, of course, to discover a means for its prevention as well as for its control. It is an involved problem, going much deeper than local manifestations. A systematic and comprehensive study of the problem was made possible at this School, in February, 1930, through the generous aid of the Commonwealth Fund. The special Caries Research Advisory Committee and the special Caries Research Staff of the School divided the problem into five phases: biochemical, bacteriological, histological and pathological, animal experimentation, and clinical experimentation. This is a presentation of the preliminary phase of our clinical (human study) work from February, 1930, to November, 1931.

Much work had formerly been done by others, and a great deal written on researches in this field. In order to discover what was authoritative and pertinent, much of the literature on this phase was reviewed before experimentation was begun. It was evident that metabolism plays a prominent and effective rôle in establishing immunity or susceptibility to dental caries. Further, study of the

[1] A preliminary report was included in the proceedings of the tenth general meeting of the International Association for Dental Research, Columbus, O., March 19 and 20, 1932; *Journal of Dental Research*, 1932, *12*, p. 538.

relation of dental caries to vitamin requirements, while not necessarily listing dental caries as a specific nutritional disorder, has led some workers to class it among the "deficiency" diseases. None of the dietary studies to date, however, clearly indicates whether the influence of diet is on the environmental factors or on the ability of the tooth structure to resist the attack of the environmental factors. Each view has its advocates; it is possible that the sum of the dietary factors may function in both the ways cited above.

It seemed desirable, then, first to observe the effect of a well balanced so-called "protective" diet on the teeth of children in which caries was already present. If such observations indicated a nutritional control in operation, the next step, obviously, would be to analyze and evaluate its makeup. The results reported by Drain, Boyd, and Nelson (1) led us to select, for our preliminary observations, the diet used by them. We planned to secure several groups of children, in institutions such as orphanages and boarding schools, to be kept under constant control and observation. Three orphanages in New York City were selected and permission was secured to make dental examinations of the children. The sources of the children were clearing bureaus, city courts, other institutions, and private homes. Dental examinations were made, in these three orphanages, of about one hundred sixty-five children ranging in age from five to seventeen years. It was impossible however, to segregate groups for dietary control in any of these institutions; and, because of the surprisingly low incidence of dental caries, they did not furnish material for study from the standpoint of producing or approaching immunity to dental caries by dietary means. However, the excellent general and dental health of these children, and the high standard of the dietaries and living conditions, in these orphanages indicated persuasively the probability of a close relation of diet to dental health; also the desirability of further study of this phase of the problem in a more suitable field.

After considerable searching for institutions in which it would be possible not only to observe the effect on caries prevention, but also to obtain accurate data on the nutritive elements of the diet actually eaten day by day by both control and experimental groups, three orphanages in the state near New York City were selected toward the

close of 1930. These homes are admirably organized for our purposes. They are conducted on the cottage plan, with twenty to thirty children in each cottage in charge of a cottage mother and assistant. Meals are prepared in the kitchens of the cottages according to the individual needs for each house, as compiled by the cottage mothers. Under this system it was possible to prescribe a definite menu to the group under study, to be certain that the group actually consumed the dietary articles prescribed, and to secure accurate record of the foods served and consumed. Our experiments here reported were carried out in these three homes, which are designated hereafter as Homes A, B, and C. The grouping in each home was as follows:

Home A—Experimental Group, 30 children; Control Group, 20 children.

Home B—Experimental Group, 27 children; Control Group, 11 children.

Home C—Experimental Group, 18 children; Control Group, 28 children.

The experimental and control groups in Homes A and B included boys and girls. In Home C it was not possible to have mixed groups; the experimental group consisted of 18 girls and the control of 31 boys.

The results here tabulated involved the feeding of children in three homes over a period of from six to seven months, during which time the experimental diet was contrasted with group feeding of control groups on the unsupplemented basal diet of these homes. These basal diets, of a high standard, were devised on recognized principles of nutrition; hence the contrast between the experimental groups and the control groups is less than might otherwise have been developed. In Homes A, B, and C our supplementing aimed to provide the conditions suggested by Boyd, Drain and Nelson (1). This consisted in building up the basal diet for the children in the experimental group to insure the following ration of "protective" foods per day:

One quart of milk
One or two eggs
One serving of meat, fish, chicken or liver
Two vegetables ($\frac{1}{2}$ cup considered as a medium serving)
One orange or apple or tomato
One fruit besides the above

Six teaspoons of butter
One teaspoonful of cod-liver oil

These are the factors Boyd *et al.* suggest as caries-preventive. In these tests this program was carried out so far as practicable, with one modification. The cod-liver oil was given in doses of three teaspoonfuls instead of one.

In addition to the above general dietary experiments in Homes A, B, and C, we also conducted an experiment on four groups of children in Home L, a state institution for mentally defective children, to determine, if possible, the specific relation of vitamin C to the control of dental caries. Only high grade types of children were selected, and their diagnoses carefully noted. Any diagnosed organic pathology that might conceivably interfere with normal metabolism rendered the child ineligible for either the experimental or the control groups. The first dental examinations were made in February, 1931. Four experimental groups of sixteen each, and one large control group of eighty-eight, were selected. The administration of vitamin C was begun March 9, 1931, as follows (all groups receiving the orphanage diet):

(1) Control Group: orphanage diet alone
(2) Experimental Group I: plus the juice of three oranges and three ripe bananas daily
(3) Experimental Group II: plus eight ounces of orange juice daily
(4) Experimental Group III: plus three bananas daily
(5) Experimental Group IV: plus six ounces of tomato juice daily

This supplementing aimed at a daily increase in vitamin C, but obviously also affected other vitamin, mineral, and nutrient proportions in the diet.

The dental examinations in all the homes were made as thorough as possible. Any abnormal condition of the gums, no matter how slight, was recorded as to its degree of severity. The teeth were carefully examined as to the character and color of the enamel, stains, cleanliness, abnormal conditions, and caries. All cavities were noted and recorded on special charts. Any fissure deep enough to catch the explorer was reported as a cavity; these were carefully noted. The compound cavities were also outlined on the charts according to their location and involvement of tooth structure.

TABLE 1

Data relating to School A: Analysis of daily menus to show comparative intakes of nutrients

Control group

DAYS	FOOD EATEN	PROTEIN	FAT	CARBO-HYDRATE	CALO-RIES	CALCIUM	PHOS-PHORUS	IRON
	Gms.	*Gms.*	*Gms.*	*Gms.*		*Gms.*	*Gms.*	*Mgms.*
Sunday	1150	59	120	272	2404	0.971	1.065	8.9
Monday	1342	70	85	284	2181	0.833	1.034	8.8
Tuesday	1245	68	54	278	1870	0.864	1.054	9.6
Wednesday	1348	65	106	282	2342	0.977	1.082	13.3
Thursday	1395	68	119	347	2731	1.166	1.171	7.4
Friday	1365	75	81	217	1897	1.413	1.430	8.9
Saturday	1278	92	113	284	2521	1.194	1.549	12.7
Total	9124	497	678	1964	15946	7.418	8.385	69.6
Daily average	1303	71	97	281	2292	1.060	1.198	9.9

Exper. Group I

Sunday	1610	65	65	203	1657	1.061	1.281	10.3
Monday	2353	84	100	249	2232	1.034	1.289	14.4
Tuesday	1610	84	85	251	2105	1.359	1.439	14.8
Wednesday	1921	80	96	276	2288	1.316	1.507	13.4
Thursday	1815	77	63	326	2179	1.231	1.325	9.8
Friday	1682	72	85	260	2093	1.024	1.389	12.1
Saturday	1333	74	68	288	2050	1.073	1.167	11.5
Total	12124	536	562	1853	14604	8.198	9.397	86.3
Daily average	1732	76	80	265	2086	1.171	1.342	12.3

Exper. Group II

Sunday	1323	72	101	284	2333	1.121	1.296	10.5
Monday	1495	73	56	225	1696	0.996	1.377	11.6
Tuesday	1538	65	99	316	2425	0.996	1.121	8.8
Wednesday	1197	75	75	251	1979	1.367	1.459	11.4
Thursday	1405	55	110	195	1990	0.901	0.983	10.2
Friday	1625	74	69	220	1797	1.319	1.567	16.9
Saturday	1488	78	75	292	2155	1.064	1.257	12.7
Total	10071	492	595	1783	14375	7.764	9.060	82.1
Daily average	1453	70	87	255	2053	1.109	1.295	11.7
Average of two experimental groups	1593	73	84	260	2070	1.140	1.318	12.0

In preparing the charts to show the differences in the groups studied, the method has been as follows: Menus and quantities of food eaten per child (averages) were collected for one week. By averaging these figures we arrived at the typical approximate daily consumption per child shown in the tabulations. Thus, *tables 1*, *2*, and *3* indicate how

TABLE 2

Data relating to School B: Analysis of daily menus to show comparative intakes of nutrients

DAYS	FOOD EATEN	PROTEIN	FAT	CARBO-HYDRATE	CALORIES	CALCIUM	PHOSPHORUS	IRON
	Gms.	*Gms.*	*Gms.*	*Gms.*		*Gms.*	*Gms.*	*Mgms.*
Control group								
Monday	1462	63	95	279	2223	1.154	1.163	6.6
Tuesday	1270	76	104	305	2460	1.108	1.346	11.8
Wednesday	1242	89	77	253	2061	0.744	1.315	13.0
Thursday	1296	58	59	199	1559	1.022	1.125	11.5
Friday	1684	86	72	226	1896	0.951	1.505	11.5
Saturday	1526	84	88	309	2364	1.194	1.355	13.4
Sunday	1544	72	77	259	2017	1.002	1.1253	10.1
Total	10024	528	572	1780	14580	7.175	9.062	77.9
Daily average	1432	75	81	254	2083	1.025	1.294	11.1
Experimental group								
Monday	1912	85	96	252	2212	1.8060	1.794	11.1
Tuesday	1687	70	79	249	1987	1.106	1.424	12.2
Wednesday	1652	68	71	221	1785	1.107	1.340	10.7
Thursday	1677	64	72	248	1896	1.233	1.117	12.7
Friday	1842	73	72	239	1896	1.320	1.481	11.0
Saturday	1313	60	56	231	1668	0.960	1.190	9.6
Sunday	1618	72	58	335	2150	1.099	1.410	14.7
Total	11701	492	504	1775	13594	8.621	9.856	82.0
Daily average	1671	70	72	253	1942	1.231	1.408	11.7

the daily menus were analyzed to show comparative intakes of nutrients in Homes A, B, and C. To indicate the vitamin differences, it was necessary to have a quantitative basis for comparison. Such assays are incomplete at present. They are also subject to variation with foodstuffs from different localities, method of table preparation, etc. But with all these limitations, the values assigned in *tables 4* and *5* are at least approximately correct, and based on actual assays in

responsible laboratories. *Table 6* summarizes the data obtained by tooth examinations in Homes A, B, and C, and also shows the characteristic dietary differences between the experimental and control groups in these homes. *Fig. 1* shows graphically the characteristic differences expressed in percentage increase or decrease of experimental groups over control groups. *Tables 7* and *8*, and *fig. 2*, present

TABLE 3

Data relating to School C: Analysis of daily menus to show comparative intakes of nutrients

DAYS	FOOD EATEN	PROTEIN	FAT	CARBOHYDRATE	CALORIES	CALCIUM	PHOSPHORUS	IRON
	Gms.	*Gms.*	*Gms.*	*Gms.*		*Gms.*	*Gms.*	*Mgms.*
Control group								
Sunday	1426	75	162	333	3090	1.259	1.262	9.8
Monday	1839	76	67	288	2059	1.210	1.463	13.4
Tuesday	1542	71	107	304	2463	1.149	1.121	6.6
Wednesday	1532	93	133	335	2909	1.419	1.708	12.8
Thursday	1324	66	93	240	2061	1.144	1.288	10.5
Friday	1388	74	62	255	2074	0.780	1.056	10.3
Saturday	1444	95	116	288	2576	1.004	1.470	11.9
Total	10495	560	740	2043	17232	7.965	9.458	75.3
Daily average	1499	80	106	292	2461	1.138	1.351	10.7
Experimental group								
Sunday	1383	81	155	307	2947	1.138	1.267	9.9
Monday	1388	70	162	273	2830	1.043	1.176	4.1
Tuesday	1655	70	96	261	2188	1.045	1.200	9.1
Wednesday	1965	98	120	330	2792	1.960	1.922	49.4
Thursday	1192	97	106	282	2470	0.952	1.531	15.5
Friday	1962	97	130	314	2814	1.462	1.765	13.3
Saturday	1463	59	82	345	2354	0.874	1.053	12.3
Total	10608	572	851	2112	17995	8.474	9.894	13.6
Daily average	1515	81	121	301	2571	1.210	1.413	16.2

data similarly obtained and classified for the five groups studied at Home L.

It is shown that while differences were not large, there was, consistently, in Homes A, B, and C, less caries in the experimental groups. In Home L it is doubtful whether the differences are significant. The problem, therefore, is first to see what dietary changes are correlated

TABLE 4

Distribution of food values: School A

FOODS	APPROXIMATE VITAMIN UNITS				EXPERIMENTAL					CONTROL				
	A	B	C	D	Food eaten Gms.	V. units eaten				Food eaten Gms.	V. units eaten			
						A	B	C	D		A	B	C	D
	Gms.	*Gms.*	*Gms.*	*Gms.*										
Cereals*	...	0.5	...	...	23	...	46.0	...	...	22	...	44.0		
Bread:														
Toast and rolls		...	...	...	91	...	...	...	...	175				
Dairy products:														
Butter	0.028	...	...	7.5	60	2140.0	...	...	8	73	2605.0	...	...	9.7
Milk	0.40	3.0	28	25.0	547	1370.0	182.0	19.0	22	480	1200.0	160.0	17.0	19.2
Cheese	0.04	3.0	...	...	3	75.0	1.0	...	...	4	100.0	1.3		
Egg	0.04	2.0	...	5.0	50	1250.0	25.0	...	10	14	350.0	7.0	...	2.8
Meats:														
Beef	0.0	3.0	...	...	16	...	5.3	...	...	11	...	3.6		
Lamb	0.0	3.0	...	...	7	...	2.3	...	...	3	...	1.0		
Fish	?	4.0	...	...	0	...	...	...	...	0	...	...		
Liver	1.0	1.0	...	...	0	...	...	...	...	0	...	...		
Fruits:														
Apple	2.0	3.0	20	...	21	10.5	7.0	1.05	...	37	18.5	12.3	1.85	
Apricot	0.50	?	10	...	20	40.0	?	2.0	...	7	14.0	?	0.70	
Banana	0.50	8.0	5	...	25	50.0	3.0	5.0	...	0				
Grapefruit	5.0	5.0	2	...	0	...	...	...	...	0				
Jam: strawberry	3.0	?	10	...	0	...	...	...	...	20	9.3	?	2.0	
Orange	5.0	3.0	2	...	50	10.0	16.0	25.0	...	29	6.0	9.6	15.0	
Peach	0.50	3.0	5	...	3	6.0	1.0	0.6	...	0				
Plum	2.0	3.0	20	...	0	...	...	...	...	0				
Prune	0.10	0.8	...	...	12	120.0	1.5	0.0	...	0				
Pear	2.0	3.0	20	...	7	3.5	2.3	0.35	...	14	7.6	5.0	0.70	

Pineapple	3.0	3.0	8	...	7	2.5	0.8	0.9	...	0				
Tangerine	5.0	3.0	2	...	0	...	...	...	...	0				
Tomato	0.075	3.0	3	...	21	280.0	7.0	7.0	...	0				
Tomato juice	0.15	3.0	4	...	43	286.0	14.3	11.0	...	0				
Vegetables:														
Beet	6.0	...	...	...	7	1.0	...	...	...	12	2.0			
Bean: string	0.15	3.0	15	...	7	46.0	2.3	0.48	...	0				
Bean: kidney	2.0	2.0	...	...	7	3.5	3.5	0.0	...	0				
Carrot	0.025	3.0	15	...	12	480.0	4.0	0.90	...	57	2280.0	19.0	4.0	
Cabbage: cooked	8.0	2.0	20	...	20	2.5	10.0	1.25	...	7	1.0	3.5	0.35	
Cauliflower	5.0	2.0	20	...	12	2.4	6.0	0.60	...	12	2.4	6.0	0.60	
Celery	5.0	2.0	10	...	12	2.4	6.0	1.20	...	7	1.6	3.5	0.70	
Kale	8.0	2.0	20	...	0	...	...	...	...	0				
Lettuce	0.05	2.0	20	...	100	2000.0	50.0	5.0	...	30	600.0	15.0	1.50	
Onion	5.0	5.0	10	...	2	0.4	0.2	0.20	...	0				
Parsnip	4.0	3.0	15	...	15	4.0	5.0	1.0	...	0				
Pea	0.075	4.0	10	...	23	306.0	5.7	2.30	...	0				
Potato	4.0	3.0	20	...	100	25.0	8.3	5.0	...	114	28.0	38.0	5.70	
Spinach	0.025	1.0	10	...	35	1400.0	35.0	3.50	...	12	480.0	12.0	1.20	
Squash	?	?	?	...	0	...	...	...	...	12				
Succotash	?	?	?	...	0	...	...	...	...	0				
Turnip	4.0	3.0	15	...	0	...	...	...	...	0				
Watercress	?	?	10	...	0	...	...	...	...	0				
Cod-liver oil	0.002	...	...	0.075	12	6000.0	...	...	173	0				
Total					1370	16,160	451	93	213	1152	7705	341	51	32

* Cornmeal, corn flakes, crackels, farina, malt breakfast-food, oatmeal, ralston, wheatena, shredded wheat, etc.

TABLE 5

Distribution of food values: Schools B and C[1]

FOODS	SCHOOL B								SCHOOL C							
	Experimental				Control				Experimental				Control			
	Food eaten Gms.	V. units eaten			Food eaten Gms.	V. units eaten			Food eaten Gms.	V. units eaten			Food eaten Gms.	V. units eaten		
		A	B	C		A	B	C		A	B	C		A	B	C
Cereals*	28	...	56.0	...	23	...	46.0	...	30	...	60.0	...	26	...	52.5	...
Bread:																
Toast and rolls	180	...	...	...	190	...	...	...	171	...	...	...	177			
Dairy products:																
Butter	80	2860.0	...	...	56	2000	...	...	60	2140.0	...	...	47	1680.0		
Milk	720	1800.0	240.0	25.0	480	1200	160.0	17.0	480	1200.0	160.0	17.0	480	1200.0	160.0	17.0
Cheese	0	...	...	...	0	...	...	...	0	...	...	...	0			
Egg	50	1250.0	25.0	...	21	520	10.50	...	50	1250.0	25.0	...	14	350.0	7.0	
Meats:																
Beef	23	...	7.7	...	3	...	1.0	...	12	...	4.0	...	7	...	2.30	
Lamb	0	...	...	...	3	...	1.0	...	0	...	...	...	3	...	1.0	
Fish	4	...	1.0	...	3	...	0.75	...	3	...	0.75	...	3	...	0.75	
Liver	0	...	...	...	3	3	3.0	...	2	2.0	2.0	...	0	...	...	
Fruits:																
Apple	43	21.5	14.3	2.15	30	15	10.0	1.50	43	21.50	14.30	2.15	30	15.0	14.30	2.15
Apricot	8	16.0	?	0.80	0	...	...	...	0	...	...	...	12	60.0	?	1.20
Banana	12	24.0	1.5	2.40	30	60	3.30	6.0	12	24.0	1.50	2.40	12	24.0	1.50	2.40
Grapefruit	12	2.4	2.4	6.0	0	...	...	...	0	...	...	...	0			
Jam: strawberry	0	...	...	...	0	...	...	...	30	10.0	?	3.0	0			
Orange	57	11.4	19.0	29.0	30	6.0	10.0	15.0	12	2.50	4.0	6.0	14	3.0	3.0	7.0
Peach	6	12.0	2.0	1.2	30	60.0	10.0	6.0	12	2.40	4.0	2.40	0			
Plum	0	...	...	...	0	...	...	...	12	6.0	4.0	0.60	12	6.0	4.0	0.60
Prune	12	120.0	15.0	0.0	12	120.0	15.0	...	21	210.0	26.50	...	14	140.0	17.0	
Pear	12	6.0	4.0	0.6	0	...	...	...	30	15.0	10.0	1.50	12	6.0	4.0	0.60

Pineapple	12	14.0	4.0	1.5	12	14.0	4.0	1.50	0	...	...	...	0			
Tangerine	0	...	...	...	12	2.4	4.0	6.0	0	...	...	...	0			
Tomato	30	400.0	10.0	10.0	0	...	...	...	0	...	...	...	30	400.0	10.0	10.0
Tomato juice	0	...	...	...	0	...	...	...	0	...	...	...	0			
Vegetables:																
Beet	7	1.1	...	...	15	2.5	...	...	0	...	...	...	18	3.0		
Bean: string	0	...	...	...	0	...	...	...	15	100.0	5.0	1.0	15	100.0	5.0	1.0
Bean: kidney	0	...	...	...	0	...	...	...	0	...	...	...	0			
Carrot	15	600.0	5.0	1.0	3	120.0	1.0	0.20	7	280.0	3.50	0.50	0			
Cabbage: cooked	15	2.0	7.5	0.75	7	1.0	3.50	0.35	43	5.45	14.30	2.15	15	2.0	5.0	0.75
Cauliflower	0	...	...	...	0	...	...	...	0	...	...	...	0			
Celery	30	6.0	15.0	3.0	30	6.0	15.0	3.0	21	4.20	10.50	2.10	0			
Kale	0	...	...	...	0	...	...	...	0	...	...	...	15	2.0	7.5	0.75
Lettuce	85	1700.0	42.5	4.25	43	860.0	21.50	2.15	100	2000.0	50.0	5.0	0			
Onion	0	...	...	...	19	4.0	3.75	1.90	0	...	...	...	18	4.0	3.6	1.80
Parsnip	0	...	...	...	0	...	...	...	0	...	...	...	0			
Pea	15	200.0	4.0	1.50	25	333.0	6.35	2.50	15	200.0	4.0	1.50	33	444.0	8.0	3.30
Potato	57	14.0	19.0	2.85	71	18.0	23.60	3.55	107	27.0	36.0	5.35	125	31.0	42.0	6.0
Spinach	15	600.0	15.0	1.50	15	600.0	15.0	1.50	0	...	...	...	15	600.0	15.0	1.50
Squash	0	...	...	...	0	...	...	...	15	...	...	...	0			
Succotash	20	...	...	...	15	...	...	...	0	...	...	...	0			
Turnip	0	...	...	...	0	...	...	...	15	4.0	5.0	1.0	0			
Watercress	0	...	...	...	3	...	...	0.30	0	...	...	...	0			
Cod-liver oil	12	6000.0	...	...	0	...	...	...	12	6000.0	...	...	0			
Total	1474	15,660	510	94	1184	5945	368	68	1330	13,504	444	54	1147	5070	364	56

[1] The approximate vitamin-D units in the dairy products and cod-liver oil for these two schools were these: *School B: Exp.*—butter 10.7; milk, 29.0; eggs, 10.0; cod-liver oil, 173; total, 223; *Control*—butter 7.5; milk, 19.0; eggs, 4.2; total, 31. *School C: Exp.*—butter, 8; milk, 19; eggs, 10; cod-liver oil, 173; total, 210; *Control*—butter, 6.3; milk, 19.0; eggs, 2.8; total, 28.

* Cornmeal, corn flakes, crackels, farina, malt breakfast-food, oatmeal, ralston, wheatena, shredded wheat, etc.

TABLE 6

Data obtained by tooth examinations in Homes A, B, and C; and summaries of characteristic dietary differences between the experimental and control groups

Orphanage	A			B			C		
Dental data									
Group	Ex.	Con.	Diff.	Ex	Con.	Diff.	Ex.	Con.	Diff.
			%			%			%
Children examined	30	20		27	11		18	28	
Teeth examined	766	520		622	286		427	644	
Teeth carious	256	187		177	69		128	214	
Carious surfaces	387	287		270	96		194	324	
Percent carious	10.1	11.03		8.68	6.71		9.09	10.06	
Percent increase during experimental period	0.13	0.82		0.26	0.35		0.29	1.91	
Increase of control over experimental	×6.3			×1.35			×6.6		
Dietary data									
Food eaten (gms.)	1370.0	1152.0	19	1474.0	1184.0	26.0	1330.0	1147.0	14
Protein (gms.)	73.0	71.0		70.0	75.0		81.0	80.0	
Protein calories	292.0	284.0	3	280.0	300.0	−7.0	324.0	320.0	1
Total calories	2070.0	2292.0	−10	1942.0	2083.0	−7.0	2571.0	2461.0	−4
Protein calories (%)	14.1	12.4		14.40	14.4		12.6	13.0	
Calcium (gms.)	1.14	1.060	8	1.231	1.025	2.0	1.210	1.138	6
Phosphorus (gms.)	1.318	1.198	10	1.408	1.294	9.0	1.413	1.351	4
Ca/P ratio	0.86	0.88	−2	0.88	0.80	10.0	0.86	0.84	2
Iron (mgms.)	12.0	10.0	20	12.0	11.0	9.0	16.0	11.0	45
Carbohydrate (gms.)	292.0	281.0	4	253.0	254.0	−0.4	301.0	292.0	3
Fat (gms.)	84.0	97.0	−13	84.0	97.0	−13.0	121.0	106.0	14
Milk (gms.)	547.0	480.0	14	720.0	480.0	50.0	480.0	480.0	0
Egg (gms.)	50.0	14.0	257	50.0	21.0	138.0	50.0	14.0	257
Fruit (gms.)	209.0	107.0	95	204.0	156.0	30.0	172.0	136.0	26
Vegetables (gms.)	352.0	263.0	33	274.0	232.0	18.0	329.0	251.0	31
Cod-liver oil (gms.)	12.0	0.0		12.0	0.0		12.0	0.0	
Vitamins (units):									
A (U.S.P.)	16,160	7705	110	15,660	5945	163	13,504	5097	165
B (Sherman)	451	341	32	510	368	38	444	364	12
C (Sherman)	93	51	82	94	68	38	54	56	−3
D (Steenbock)	213	32	558	223	31	620	210	28	650

TABLE 7

Data obtained by tooth examinations in Home L; and summaries of characteristic dietary differences among the groups (vitamin-C series)

Groups	Control	I		II		III		IV	
Dental data									
Nature of supplement	None	3 oranges 3 bananas	Diff.	6 oranges	Diff.	3 bananas	Diff.	6 oz. tomato juice	Diff.
			%		%		%		%
Children examined	84	16		16		16		16	
Teeth examined	2143	415		411		416		408	
Teeth carious	472	131		122		104		92	
Carious surfaces	713	225		254		173		133	
Percent carious	6.65	10.84		12.36		8.31		6.51	
Percent increase during experimental period	1.0	1.73		0.86		1.06		0.92	
Increase or decrease of control over experimental			73		−14		6		−8
Dietary data									
Food eaten (gms.)	644.0	1218.0	89	868.0	35	1106.0	56	812.0	26
Protein (gms.)	77.0	84.0		79.0		83.0		78.0	
Protein calories	308.0	336.0	9	316.0	3	332.0	8	312.0	1
Total calories	2285.0	2797.0	22	2395.0	5	2742.0	20	2348.0	3
Protein calories (%)	13.5	12.0		13.20		12.10		13.30	
Calcium (gms.)	0.503	0.576	14	0.567	12	0.544	8	0.513	2
Phosphorus (gms.)	0.888	1.049	18	0.924	4	1.031	16	0.915	3
Ca/P ratio	0.57	0.54	−5	0.61	7	0.53	−7	0.56	−2
Iron (mgms.)	7.80	8.3	6	8.20	5	8.10	4	?	
Carbohydrate (gms.)	285	399.0	45	310.0	9	387.0	36	292.0	2
Fat (gms.)	93	96.0	3	93.40	0	96.0	3	96.40	3
Milk (gms.)	120	120.0		120.0		120.0		120.0	
No egg									
Fruit (gms.)	30	604.0		254.0		492.0		198.0	
Vegetables (gms.)	169	169.0		169.0		169.0		169.0	
No cod-liver oil									
Vitamins (units):									
A (U.S.P.)	2829.0	3779.0	33	2874.0	16	3757.0	31	3949.0	40
B (Sherman)	231.0	326.0	41	306.0	32	289.0	25	287.0	24
C (Sherman)	25.0	173.0	590	137.0	448	117.0	367	67.0	168
D (Steenbock)	9.6	9.6		9.6		9.6		9.6	

TABLE 8

Dietary data relating to Home L (vitamin-C series); four groups

Diet common to all groups

FOODS	FOOD EATEN	VITAMIN UNITS EATEN			
		A	B	C	D
	Gms.				
Breakfast cereals	63	...	126.0		
Bread toast and rolls	180				
Dairy products:					
Butter	35	1250.0	...	...	4.6
Milk	120	300.0	40.0	4.0	5.0
Cheese	7	175.0	2.3		
Meats:					
Beef	37	...	12.3		
Fish	4	...	1.0		
Fruits:					
Apples	8	4.0	2.6	0.40	
Tomato	21	280.0	7.0	7.00	
Vegetables:					
Beans: string	25	166.0	8.0	1.66	
Beans: baked	3	6.5	6.5		
Carrots	16	640.0	5.3	1.06	
Cabbage: cooked	25	3.0	12.5	1.25	
Cabbage: raw	8	1.0	4.0	8.0	
Corn	50	?	?	?	
Onions	16	3.0	3.0	1.60	
Squash	16	?	?	?	
Total	644	2829	231	25	9.6

Addenda to different groups

Group I:					
Juice of 3 oranges	112	22	37	56	
3 bananas	462	928	58	92	
Total	574	950	95	148	
Total: Gr. I	1218	3779	326	175	
Group II:					
8 ozs. orange juice	224	45	75	112	
Total: Gr. II	868	2874	306	137	
Group III:					
3 bananas	462	928	58	92	
Total: Gr. III	1106	3757	289	117	
Group IV:					
6 ozs. tomato juice	168	1120	56	42	
Total: Gr. IV	812	3949	287	67	

with the improvement in the experimental groups, and their relation to existing theories as to how dietary control functions. To make such comparison it is first necessary to review briefly some of these current theories, which have recently been summarized by Bunting (2), Hanke (3), Mellanby (4), and others. The views of McCollum and Klein (5) and the work of Hoppert, Webber, and Canniff (6), on phosphorus, stimulated the work described in this report. These articles, which have been given special attention, are briefly summarized below.

According to the older, so-called "Miller" theory, caries is the result of attack by aciduric or acidogenic organisms that occur in the mouth and are attached to the tooth surface. There is still doubt as to the species of these organisms, but in general they are believed to produce effects in two ways; first, by elaboration of an acid that dissolves enamel and, second, by elaboration of enzymes that break down the organic matrix of enamel and dentine. The customary measures of oral hygiene are based on this assumption. It is the basis of the slogan: "A clean tooth never decays." This theory fails, however, to cover the variations in tooth behavior toward attacking organisms. Dr. Alfred S. Walker of New York City, in a recent discussion, called attention to the fact that the incisors and canines of the lower human jaw frequently resist caries long after all the other teeth in the mouth have succumbed to attack. The decay of one of a pair of teeth contiguous to a crevice rich in bacteria, and the immunity of the other, is a fairly frequent observation. Teeth, in other words, show differences in resistance to bacterial attack. The increasing number of observations that correlate dietary measures with reduction in caries suggest various hypotheses as to how this immunity is produced:

(A) Dietary factors may function by reducing the attacking forces. (Effect on the environmental conditions surrounding the tooth.)

(B) Dietary factors may function by providing a circulatory condition in the tooth, and a lymph on the tooth tissues, which neutralize the forces attacking the enamel—Bödecker theory (7).

(C) Dietary factors may modify tooth structure, making it more resistant to attacking forces.

(D) Dietary factors may function in all of these ways; and the resulting immunity may be a response to a combination of rôles rather than to any specific single factor.

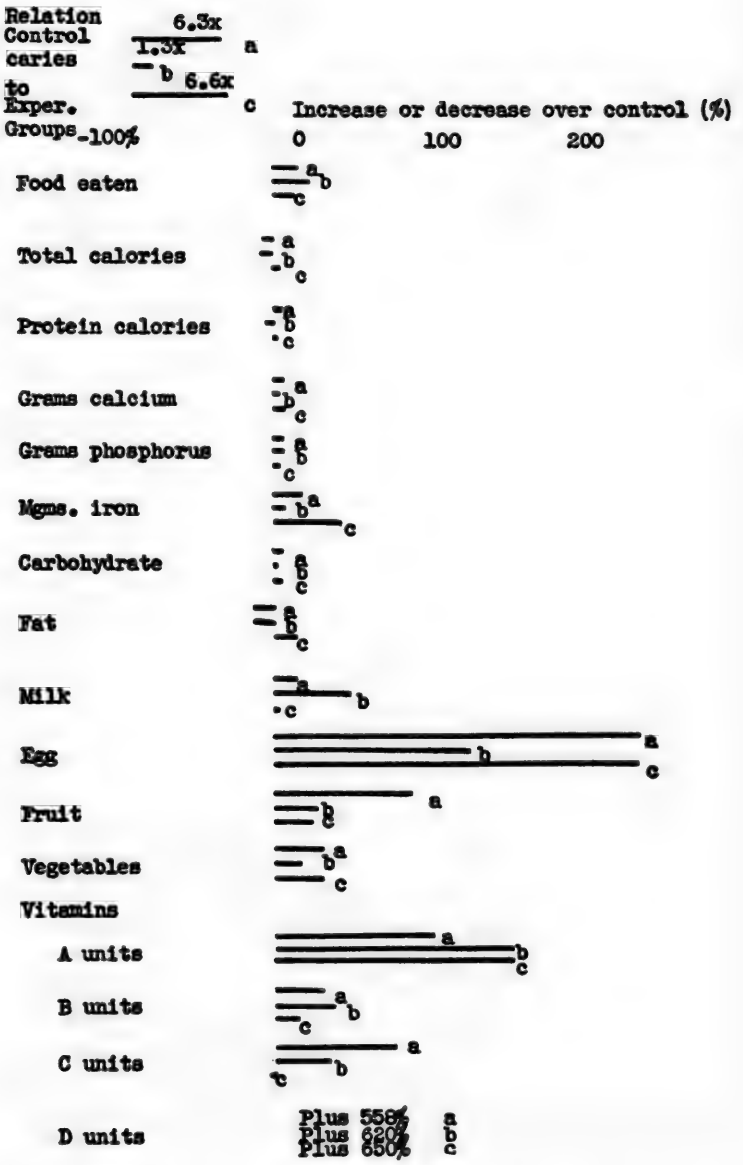


FIG. 1. Graphic presentation of differences between experimental and control-group diets in New York orphanages. (Homes A, B, and C.) Letters a, b, and c indicate orphanage groups.

In the attempt to explain the rôles of specific factors, different workers have stressed the following views.

Bunting (2) believes that dietary selection accomplishes reduction of

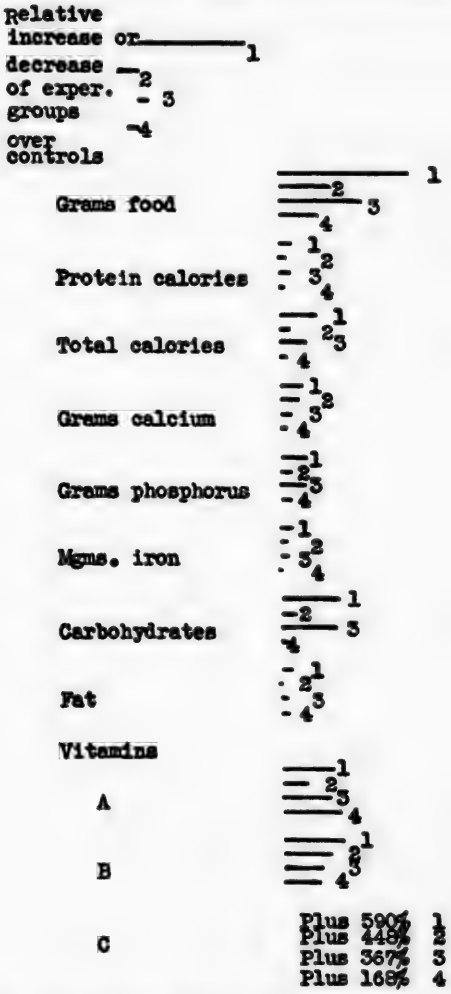


FIG. 2. Vitamin-C supplementary series. (Home L.) Data opposite horizontal lines, beginning with "Grams food," represent percentage differences in dietary factors in experimental groups contrasted with control group.

the aciduric organisms and their attachment to enamel surfaces; that a change in diet which reduces its sugar and cereal-starch content makes for such amelioration of the tooth's environment. He finds little or no evidence of immunity being accomplished by structural changes in the tooth, or by a circulating dental lymph.

Hoppert, Webber, and Canniff (6) contrasted among other factors the effect on rats of whole corn vs. sifted corn in otherwise similarly constituted diets, and found caries producible with the whole corn and not with the sifted corn. They attribute the caries incidence in the former group to retention on the molars of large particles of corn, resulting in fermentation, and in the dissolution of tooth enamel. They say: "Attempts to prevent decay of the teeth by the addition of cod-liver oil or orange juice, supplying liberal amounts of vitamins A, D, and C, or by fortifying the ration with calcium carbonate or tricalcium phosphate have been of no avail. In view of these results it is difficult to accept the theory that dental caries is due primarily to a vitamin or mineral deficiency of the diet. The results rather are in strong support of a not-at-all-modern point of view; namely, that the cause of dental decay is the decomposition, most likely by aciduric and acidogenic bacteria, of certain foodstuffs held by the teeth in some way or other."

It is typical of the present uncertainty in interpretation of dietary experiments that the data published by Hoppert *et al.* supply to Klein and McCollum (5) an entirely different explanation of the reduction in caries. They examined the phosphorus and calcium composition of the two diets, and conclude that, with a calcium intake of 0.3424 grams per 100 grams of the total diet, rat rations containing less than 0.4802 grams of phosphorus per 100 grams of diet induce caries, and that an increase in phosphorus to 0.5282 or more grams per 100 grams of diet produces immunity. This relation of phosphorus to caries is also reflected, according to them, in the phosphorus content of the blood. They say: "Caries arises in rats whose blood phosphorus falls below a critical level (about 10.5 $\pm$ 0.5 mg. of phosphorus per 100 grams of serum), while those rats whose blood phosphorus concentration is 10.5 $\pm$ 0.5 mg. or above, are immune from dental caries." Since control of blood phosphate involves adequacy of vitamin D in the diet, and perhaps of other vitamins and digestive

factors, Klein and McCollum's views of the rôle of phosphorus do not exclude these collateral factors, as they indicate in their discussion.

The fact that a deficiency of vitamin C produces profound changes in the behavior of the odontoblasts and the structure of dentine has been demonstrated by many workers. It is also a factor in the permeability of blood vessels and control of hemorrhage. These facts have led to the feeling that a deficiency of this factor might be a predisposing cause of gingivitis, periodontoclasia, and of caries. Howe was one of the first in this country to relate vitamin-C deficiency to tooth abnormality. With Wolbach he worked out a theory of vitamin-C function. Scandinavian workers, notably Höjer and Gosta Westin, have given much attention to the rôle of this factor. More recently Hanke (3) has adduced evidence, based on liberal use of orange juice (a rich vitamin-C source), that appeared to support the view that high content of vitamin C is a preventive of gum defects and caries incidence. Eddy's laboratory work in collaboration with Dalldorf and Zall has supported the view that normal dentine demands much more vitamin C in diet than is necessary to prevent macroscopic symptoms of scurvy. There are still lacking, however, data indicating direct correlation of caries immunity and vitamin-C content of diet. Hanke himself is in doubt as to whether his orange-juice results are due solely to vitamin C or to other factors in citrus-fruit juices.

In England the cumulative studies by Mellanby (4) have led her to stress vitamin D as the significant factor. She reports results such as those in *table 9*. She comments on these results as follows: "Making due allowance for the difference in the conditions of the Sheffield and Birmingham investigations, the results are sufficiently alike to be regarded as confirmatory of one another in demonstrating a great retardation of the progress of caries in those groups receiving cod-liver oil and vitamin D, when compared with the other groups. The facts indeed seem to warrant the conclusion that the incidence and rate of progress of caries in teeth, either fully or nearly calcified, can be considerably retarded by the addition of fat-soluble vitamins (A and D) to the normal diet. The general similarity of the result obtained from the use of cod-liver oil and radiostrol (viosterol in U. S. A.), respectively, suggests strongly that the potent factor in securing this retardation is vitamin D, inasmuch as it is common to both substances."

Recently, in Eddy's laboratory, Kellogg, working in collaboration with Bödecker, has found it possible not only to produce a caries of rat molars by feeding dry lemon pulp (pH 3.5), but also to avert the caries production by combining with the pulp a high content of vitamin B_1.

What light is thrown on these theories of caries by the dietary contrasts in Homes A, B, and C? The variations in Groups A, B, and C (experimentals) are useful in evaluating dietary differences. In all these groups, as contrasted with the controls, caries was reduced. But differences in dietary factors within these same groups help to prevent overstress of certain differences. For example, while Group B decreased protein-calorie intake, A and C increased it. This change

TABLE 9

Data summarized by Mellanby (4)

GROUPS STUDIED	PERCENTAGE OF TEETH CARIOUS			PERCENTAGE INCREASE IN DEGREE OF CARIES (A.C.F.)
	First inspection	Final inspection	Increase	
Sheffield:				
Olive oil	25.96	35.96	10.00	62.13
Cod-liver oil	42.86	44.41	1.55	10.28
Vitamin D	45.13	46.14	1.01	6.57
Birmingham:				
Olive oil	15.59	23.22	7.63	45.85
Cod-liver oil	19.26	22.23	2.97	9.81
Vitamin D	22.18	24.27	2.09	10.12

could not therefore be considered significant. Let me then review each set of differences and their possible support or lack of support for theories reviewed above.

An outstanding effect of the Boyd, Drain, and Nelson diet in our series was an increase in the bulk of food eaten, with only a slight decrease in calorie intake and a slight increase in grams of carbohydrate consumed. This effect is not a contradiction of Bunting's views on carbohydrate intake as a factor, in spite of the fact that carbohydrate intake was actually increased. It reduced the cane sugar and cereal forms, and increased the fruit and vegetable forms. In so doing, it might have affected the oral flora. Our observations did not measure such bacterial changes—a point reserved for later study. It

is, however, to be noted that the tendency of fruit and milk sugars in other parts of the digestive tract is to increase rather than decrease gram-positive organisms. Arnold has recently shown this effect in rat-feeding experiments (see *table 10*). It may be that oral bacteria which attack teeth are different from the gram-positives developed in other parts of the tract, but it would seem odd if foods that produce high and low gram-positives elsewhere in the digestive tract reverse that effect in the mouth. Marriott expresses the belief that the idea that candy and other sweets harm the teeth "belongs with other fallacious theories." What sweets really do is to destroy appetite for foods essential to healthy teeth, he says. Our results, therefore, neither confirm nor contradict Bunting's views.

TABLE 10

Gram-negative to gram-positive ratios: digestive-tract regions sampled (Arnold)

SUGAR; AND PERIOD OF ADMINISTRATION AT TIME OF TEST	STOM.	DUOD.	JEJ. 1	JEJ. 2	JEJ. 3	ILEUM	CECUM	RECTUM
Sucrose; 8–12 wks.	1:1	1:6	1:4	1:6	1:6	1:5	1:2.5	1:1.5
Cornstarch; 4–6 wks.	1:4	1:1½	1:2	1:2	1:2.5	1:2.5	1.5:1	1:2
Banana pulp (ripe); 5–8 wks.	1:8	1:6	1:10	1:8	1:6	1:6	1.5:1	1.5:1
Lactose	1:30	1:31	1:35	1:30	1:44	1:44	7:8	1:6

In none of these groups was there inadequacy of calcium intake. Since we have no retention data on these cases, it is impossible to say whether utilization was better in one group or the other. In spite of this lack of data on utilization, two features of the diet suggest a possible difference here. Increase in fruit juice helps absorption of calcium. Decrease in fat also lessens the danger of elimination of unabsorbed calcium soaps in the feces. The fact that the Experimental Group, Home C, however, actually increased its fat intake over the control, and yet had the same caries reduction as the experimental Group, Home A, tends to reduce faith that variation in fat intake was significant here. The effect of fruits on calcium and phosphorus utilizations as a possible factor for success is one that should not be dismissed without consideration.

The Ca/P ratios were slightly increased in the experimental groups; but since caries was quite absent in Hoppert's studies, where this

ratio was 0.66, and in all of our groups in Homes A, B, and C the ratio was 0.80, we must conclude that the slight changes we noted had little bearing on the differences in the caries-reduction observed. It is also difficult to find in the slight phosphorus increases much support for the Klein-McCollum view as of primary importance in our differential between groups.

It is of course obvious that while increased amounts of milk, egg, cod-liver oil, fruits, and vegetables produced *small* differences between groups in calories, essential mineral salts, acid-base value, etc., they produced *very marked* differences in vitamin values, vitamins A, B, C,

TABLE 11

Data on units of vitamin C received per child in the control and experimental groups in the four homes

Homes	A		B		C		L				
Group	Ex.	C.	Ex.	C.	Ex.	C.	C.	I	II	III	IV
Units of vit. C per day	93	51	94	68	54	56	25	173	137	117	67
Caries increase (%)	0.13	0.82	0.26	0.35	0.29	1.91	1.0	1.73	0.86	1.06	0.92

TABLE 12

Data on units of vitamin B received per child in the control and experimental groups in the four homes

Homes	A		B		C		L				
Group	Ex.	C.	Ex.	C.	Ex.	C.	C.	I	II	III	IV
B units per day	451	341	510	368	444	364	231	326	306	289	287
Caries increase (%)	0.13	0.82	0.26	0.35	0.29	1.91	1.0	1.73	0.86	1.06	0.92

and D having been much increased in this series of experimental groups. If one sought confirmation of Mellanby's view as to the importance of vitamin D, our series in Homes A, B, and C would tend to support that view. It was the vitamin most tremendously increased by the diet. The increase in vitamin A is next greatest and in vitamins B and C next in order. How significant are the increases?

The studies at Home L were devised to evaluate the effect of added vitamin C in three forms: orange juice, banana, and tomato juice. If we contrast the actual number of units per child in the control and experimental groups in Homes A, B, C, and L, and the caries-produc-

tion data, we get the values in *table 11*. Certainly one cannot draw from these data support for the view that vitamin-C intake was of prime importance in the differences observed.

Recent work has shown that vitamin B has a marked effect on gastro-intestinal normality, a change in which is rapidly reflected in oral conditions. Unfortunately the available assays do not differentiate between contents of vitamins B and G, but they are an indication of the relative richness in vitamin B. If we tabulate the vitamin-B differences in our groups as we have done above with vitamin C, we get the data in *table 12*. If we arrange the data in *table 12* in the order of highest vitamin-B content to lowest, we get the following comparisons with caries incidence:

Group	*Vitamin B*	*Caries increase*
B-Ex.	510	0.260
A-Ex.	451	0.130
C-Ex.	444	0.290
B-Control	368	0.350
C-Control	364	1.91
A-Control	341	0.82
L-I	326	1.73
L-II	306	0.86
L-III	289	1.06
L-IV	287	0.92
L-Control	231	1.00

Variations in incidence of caries in the vitamin-B groups fail to show this vitamin as the significant single responsible agent. The work on vitamin C gave similar findings. The vitamin-B work does suggest, however, that total caries-incidence may be correlated to a degree with total vitamin-B intake. We get less incidence with dosages of over 400 units than with amounts below 400. When these vitamin-B figures are compared with those obtained in another connection, to establish the human requirement, it appears that 400 or better are needed for full normality of human gastro-intestinal function.

The high vitamin-D intake of the experimental groups in Homes A, B, and C would be more satisfactory support of Mellanby's views if it

were not for the variations in Groups I-IV of Home L. Here we get variation in caries incidence; and yet in these different groups the daily intake of vitamin D was small and constant.

Conclusions

(1) The results fail to demonstrate conclusively the super-importance of any one dietary factor.

(2) They tend to confirm the theory of the importance of diet as a factor in the nutritional control of dental caries.

(3) They supply a set of dietary differences correlated with reduction in caries.

(4) They tend to support Mellanby's views as to the importance of vitamin D. It was the vitamin most deficient in the regulation diets, and most tremendously increased in the experimental diet.

(5) These differences suggest opportunity for trying out the rôle of single factors, keeping others constant and adequate until the particular rôle of each variant in the process is delimited.

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EFFECT OF MEALS, AND THE CHEWING OF PARAFFIN AND GUM, ON THE ACID-NEUTRALIZING ACTION OF SALIVA

J. C. FORBES, M.A., PH.D.

Department of Biochemistry, Medical College of Virginia, Richmond, Va.

It is generally accepted that one of the important factors in the origin of dental caries is a solution of the enamel by acids produced by microörganisms in the mouth. If this is the case, and since acid-forming organisms are present in the mouth of many immune individuals, one might assume that the acid-neutralizing action of the saliva must be an important factor in determining immunity or susceptibility to the disease. It is however not agreed that this is the case. Sixteen years ago Gies (1), referring to diagnosis from superficial examination of saliva, stated: "It is biologically absurd to deduce anything of biological import from the nature and degree of reaction of any specimen and any pair of specimens from anyone under any condition." Recently Karshan, Krasnow and Krejci (2) were also unable to demonstrate any relationship between salivary pH, acid-neutralizing action, and susceptibility to dental caries. Hawkins (3), however, from an extensive series of investigations, concluded that the acid-neutralizing action of saliva is an important factor in the etiology of caries. Pickerill (4) compared the acid-neutralizing action of the saliva of fifty immune Maori children with a corresponding number of susceptible European children, and found that it was approximately six times greater in the former than in the latter.

The methods employed by these investigators are, however, open to criticism. Hawkins took the pH value of saliva, after standing for one hour, as an indication of the acid-neutralizing action. Although such a procedure may possibly lead to correct conclusions, it is evident that the pH of a solution is a measure of its neutralizing action only when it is considered in relation to the amount of buffer substances present. The alkalinity of saliva also tends to increase on standing,

owing to loss of carbon dioxide and possibly to gain of nitrogenous substances from bacterial decomposition; so that the reaction of the saliva as measured by him was by no means comparable to that naturally existing in the mouth. Karshan and co-workers, and Pickerill, titrated a known volume of the saliva with dilute acid, using methyl orange as indicator. It would seem that the main objection against such a procedure is the uncertainty of the methyl orange end-point when one is titrating a buffered solution like saliva. It has also been the present author's experience that the increase in the acid-neutralizing action of the saliva, as measured by his potentiometric method, arises primarily in the latter part of the titration. It would therefore seem that this increase is likely due to the formation, by bacterial action, of weakly-basic protein decomposition-products. Since the amount of these products is always an unknown factor, it is obviously desirable to titrate as short a distance as feasible into the pH range where they are operative.

In view of the existing uncertainty regarding the part played by the acid-neutralizing action of saliva in the etiology of dental caries, it was decided to attempt a further study of the problem, using a method somewhat different from that employed by other investigators. Since Starr (5) and Henderson and Millet (6) have shown that the salivary pH falls after a meal, it was decided to study further some of the factors influencing salivary neutralizing action, before making a comparative study of the acid-neutralizing action of caries-immune and -susceptible individuals. A series of experiments was therefore carried out on the effects of diet, of relation of meal-time to time of collection, and of such stimuli as mastication of food, gum- and paraffin-chewing, and oral administration of meat extract. In addition, a comparative study of neutralizing action, phosphorus concentration, and CO_2-combining power has been carried out in a number of cases. The results obtained on the effect of diet were presented in a preceding paper of this series.[1]

Collection of saliva. All samples of saliva, excepting those collected during mastication, were obtained with a muscular movement of the jaws that was kept as slight as possible to reduce the danger of increasing neutralizing action by undue exercise of the jaws.

[1] Forbes and Gurley: *Journal of Dental Research*, 1932, xii, p. 637; Aug.

Acid-neutralizing action was determined with quinhydrone electrodes. The electrode vessel for the unknown solution was the same as that described by Garrison, Nicholas, and Pasternack (7) for the determination of H-ion concentration with the hydrogen electrode. The details of the electrode vessel are shown in *fig. 1.* All determinations were carried out as quickly as possible after collecting the

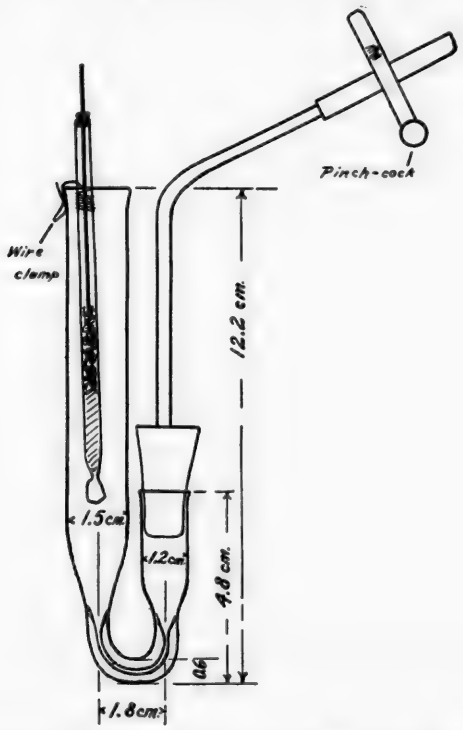

FIG. 1. DRAWING OF QUINHYDRONE-ELECTRODE VESSEL USED BY AUTHOR

sample, since the neutralizing action increases on standing. In order to approximate the condition actually existing in the mouth, expired air from the operator was bubbled through the solution until saturated, immediately before the actual pH measurement. This served also to stir the solution and saturate it with quinhydrone. The pH thus determined must be a close approximation to that present in the mouth,

TABLE 1

Effects of meals on the neutralizing action of saliva

EXP. NO.	HCl ADDED cc.	pH AND TIME OF COLLECTION							REMARKS
		7:30	8:30	9:30	10:30	12:30	1:00	3:00	
1	0	6.9	6.9	6.9	6.8	6.9	6.6	6.9	No breakfast; meat sandwich, 12:15 p.m.
	1	6.0	6.2	6.1	6.4	6.2	5.3	6.1	
	0.5	5.1	5.2	5.4	5.6	5.5	4.3	5.3	
2	0	6.8	6.9	6.9	6.9	6.9	6.7	6.9	No breakfast; peanut-butter sandwich, 12:15 p.m.
	1	6.0	6.2	6.0	6.1	6.2	5.8	6.0	
	0.5	5.0	5.4?	5.1	5.5	5.5	4.8	5.2	
3	0	6.7	6.7	6.8	6.8	6.6[1]	6.8[2]	6.8[3]	No breakfast; meat sandwich, 11:00 a.m.
	1	5.7	6.0	5.8	6.1	5.6	6.1	6.1	
	0.5	4.9	5.0	4.8	5.2	4.7	5.7	5.7	
4	0	6.6	6.8	6.7	6.8	6.8	6.3		No breakfast; bread and butter, 12:00 noon.
	1	5.8	6.3?	5.7	5.9	5.8	4.8		
	0.5	5.1	5.7?	4.9	5.2	5.1	4.2		
5	0	6.7	6.7	6.8	6.8	6.8[1]	6.5	6.7	No breakfast; beans, 11 a.m.
	1	5.7	6.1	6.2	6.1	5.7	5.4	5.9	
	0.5	4.9	5.1	5.4	5.3	4.8	4.7	5.2	
6	0	6.8	6.4	6.8	6.8	6.9			2 cheese sandwiches, 7:55 p.m.
	1	6.1	5.0	5.5	6.1	5.9			
	0.5	5.3	4.1	4.5	4.9	4.9			
7	0	6.9	6.7	6.7	6.9	7.0			Peanut-butter sandwich, 8 p.m.
	1	6.3	5.5	6.1	6.3	6.4			
	0.5	5.7	4.6	5.2	5.4	5.7			
8	0		6.6	6.8	6.9	6.8	6.8[2]	6.9[3]	Cornflakes, toast, oranges and coffee, 8 a.m.; ham sandwich, 1 p.m.
	1		5.1	5.8	6.1	6.2	5.4	6.2	
	0.5		4.7	4.8	5.1	5.3	4.7	5.2	
9	0		6.4	6.4	6.5	6.8			Milk, toast and oranges, 8 a.m.
	1		5.0	5.4	5.8	6.1			
	0.5		4.6	4.7	5.2	5.7			
10	0		6.4	6.5	6.7	6.7	6.4	6.8	Cornflakes, toast and coffee, 7 a.m.; peanut-butter sandwich, 12:45 p.m.
	1		5.1	5.7	5.8	5.9	5.3	5.8	
	0.5		4.3	4.9	4.8	4.8	4.5	4.8	
11	0		6.6	6.7	6.7	6.9	6.8	7.1	Grapenuts, oranges, toast and milk, 8 a.m.; ham sandwich, 12:45 p.m.
	1		5.2	5.7	5.8	6.1	5.7	6.3	
	0.5		4.4	4.9	4.8	5.0	5.0	5.5	
12	0		6.6	6.8	6.9	6.9	6.7	7.0	Cornflakes, oranges, toast and milk, 8 a.m.; ham sandwich, 1 p.m.
	1		5.1	5.8	6.1	6.2	5.4	6.2	
	0.5		4.7	4.8	5.1	5.3	4.7	5.3	
13	0	6.8	6.9	6.9	6.9	6.9	6.5[2]	...[3]	2 oranges, 8 a.m.; ham sandwich, 1 p.m.
	1	5.9	6.2	6.0	6.1	6.2	5.3	5.7	
	0.5	5.2	5.1	5.2	5.3	5.7	4.7	5.2	
14	0	6.8	6.8	6.9	6.8	6.9	6.9		No breakfast; 2 large oranges, 10 a.m.
	1	6.2	6.1	6.1	6.0	6.1	6.2		
	0.5	5.3	4.9	5.2	5.2	5.4	5.4		
15	0	6.8	6.8	6.9	7.0	7.0			No breakfast; 2 large oranges, 9:55 a.m.
	1	6.0	6.0	6.0	6.2	6.3			
	0.5	5.2	4.9?	5.1	5.6	5.5			
16	0	6.6	6.9	6.9	6.7	6.9			No breakfast; 2 oranges, 9:35 a.m.
	1	5.5	6.1	5.8	5.6	6.0			
	0.5	4.5	5.4	4.7	4.7	5.2			

[1] Collected at 12 noon. [2] Collected at 2 p.m. [3] Collected at 4 p.m.

TABLE 2

Effects of meals on the neutralizing action of saliva

SUBJECT	EXP. NO.	HCl ADDED cc.	pH AND TIME OF COLLECTION					REMARKS
J. C. F.	1		*11:30*	*12:30*	*1:30*	*2:30*		2 toasted egg-sandwiches, 1 p.m.
		0	6.9	7.0	6.8	6.9		
		1	6.2	6.2	5.7	6.1		
		0.5	5.8?	5.3	4.7	5.3		
W. P.	2		*11:30*	*12:00*	*1:30*	*2:40*	*4:30*	2 toasted egg-sandwiches, 1 p.m.
		0	7.0	7.0	6.8	6.9	7.0	
		1	6.3	6.5	6.3	6.5	6.4	
		0.5	5.9	6.3	5.8	6.1	6.0	
R. J.	3		*11:00*	*12:30*	*1:20*	*2:10*	*4:30*	Toast and coffee, 1:55 p.m.
		0	6.8	6.9	6.9	6.3	6.8	
		1	5.2	5.7	5.7	4.9	5.4	
		0.5	4.4	4.8	5.0	4.2	4.8	
C. P.	4		*11:15*	*1:10*	*1:45*	*2:40*	*4:30*	Toast and coffee, 1:35 p.m.
		0	6.5	6.9	6.9	6.7	6.8	
		1	5.5	6.0	6.0	5.5	5.9	
		0.5	4.6	5.3	5.1	4.4	5.1	
W. P.	5		*12:20*	*1:25*	*2:30*	*3:40*		Toast and coffee, 12:40 p.m.
		0	7.0	6.7	6.9	7.0		
		1	6.4	6.1	6.2	6.5		
		0.5	5.8	5.3	5.7	5.8		
J. C. F.	6		*12:20*	*12:40*	*2:15*	*3:00*	*4:00*	15 cc. meat extract, 12:21 p.m.; meat and vegetable sandwich, 1:45 p.m.
		0	6.7	6.7	6.5	6.5	6.7	
		1	6.2	5.6	5.2	5.2	6.0	
		0.5	5.4	4.8	4.6	4.5	5.4	
W. P.	7		*11:00*	*11:30*	*12:30*	*2:20*	*3:10*	15 cc. meat extract, 11:05 a.m.; egg sandwich, 1 p.m.
		0	6.8	6.9	6.9	6.8	7.0	
		1	6.2	6.2	6.3	5.6	6.2	
		0.5	5.8	5.7	6.1	5.3	5.6	
J. C. F.	8		*10:30*	*11:00*	*11:45*	*12:30*	*1:25*	15 cc. meat extract, 10:33 a.m.; toast and coffee, 1 p.m.
		0	6.6	6.9	6.7	6.9	6.5	
		1	5.8	5.9	5.8	5.7	5.3	
		0.5	5.0	4.8	4.7	4.6	4.3	
W. P.	9		*11:45*	*12:15*	*12:45*	*2:15*	*4:00*	15 cc. meat extract, 11:47 a.m.; meat sandwich, coffee and ice cream, 1:45 p.m.
		0	6.9	6.9	7.0	6.7	6.9	
		1	6.4	6.5	6.4	6.0	6.4	
		0.5	5.9	6.0	5.9	5.3	6.0	
J. C. F.	10		*12:10*	*1:30*	*1:50*	*2:20*	*4:30*	Meat and vegetable dinner, 1:20 p.m.
		0	6.7	6.7	6.4	6.5	6.6	
		1	5.4	6.1	4.7	5.3	5.5	
		0.5	4.7	5.3	4.2	4.7	4.8	
W. P.	11		*12:10*	*1:30*	*1:50*	*2:20*	*4:40*	Meat and vegetable dinner, 1:20 p.m.
		0	7.0	7.0	6.9	7.0	6.9	
		1	6.5	6.7	6.3	6.5	6.4	
		0.5	6.0	6.5	6.1	6.2	5.9	
W. P.	12		*11:30*	*12:25*	*1:00*	*2:00*		Meat and vegetable dinner, 12:10 p.m.
		0	7.0	7.0	6.8	6.9		
		1	6.4	6.2	6.1	6.3		
		0.5	5.8	5.3	5.3	5.7		
J. C. F.	13		*11:20*	*12:25*	*1:00*	*2:00*		Meat and vegetable dinner, 12:10 p.m.
		0	6.7	6.4	6.5	6.7		
		1	5.7	5.3	5.3	5.6		
		0.5	4.7	4.3	4.3	4.7		

but possibly may be slightly low, as the ensuing CO_2-tension is undoubtedly slightly greater than that actually present in the mouth.

TABLE 3

Effects of chewing paraffin or gum, etc., on the neutralizing action of saliva

SUBJECT	EXP. NO.	HCl ADDED cc.	pH AND TIME OF COLLECTION								REMARKS
W. P.	1		*11:40*	*11:50*	*12:15*	*12:30*	*12:50*	*1:20*	*2:20*	*2:50*	Chewed paraffin, 11:45–12:15 p.m.; sandwich, 1:50 p.m.
		0	7.0	7.2	7.2	7.2	7.2	7.0	6.7	6.7	
		1	6.5	7.0	6.9	6.6	6.5	6.4	6.2	6.1	
		0.5	6.2	6.9	6.9	6.3	6.2	5.8	5.7	5.4	
J. C. F.	2		*11:25*	*11:30*	*11:40*	*12:05*	*12:40*	*1:10*	*2:20*	*2:45*	Chewed paraffin, 11:29–12:06 p.m.; sandwich, 1:50 p.m.
		0	6.6	7.2	7.2	7.2	6.9	6.7	6.5	6.6	
		1	5.7	6.7	6.8	6.8	5.9	5.4	5.4	5.3	
		0.5	4.5	6.6	6.6	6.6	4.5	4.4	4.5	4.3	
J. C. F.	3		*10:20*	*10:32*	*10:47*	*11:00*	*11:32*	*1:00*	*2:15*	*5:00*	Chewed gum, 10:23–10:32 a.m.; meat sandwich and coffee, 1:45 p.m.
		0	6.7	7.2	6.9	6.8	6.7	6.7	6.6	6.8	
		1	5.5	6.8	5.6	6.0	5.8	5.7	5.1	5.7	
		0.5	4.6	6.4	4.8	5.2	4.8	4.8	4.4	4.9	
W. P.	4		*11:35*	*11:50*	*12:05*	*12:20*	*1:00*	*2:10*	*5:00*		Chewed gum, 11:40–11:50 a.m.; meat sandwich and coffee, 1:45 p.m.
		0	7.0	7.2	6.9	6.9	6.9	6.7	7.1		
		1	6.5	6.9	6.4	6.5	6.4	6.1	6.5		
		0.5	6.1	6.5	5.7	5.8	5.8	5.3	6.1		
J. C. F.	5		*10:15*	*10:50*	*11:20*	*12:00*	*12:45*	*1:10*	*1:55*	*2:05*	Chewed gum, 10:30–12:20 p.m.; meat sandwich, 1:35 p.m.
		0	6.6	6.9	7.0	7.1	6.9	6.9	6.9	6.7	
		1	5.5	6.1	6.5	6.4	6.0	6.0	5.5	5.6	
		0.5	4.7	5.0	6.1	5.7	5.0	5.2	4.7	4.5	
W. P.	6		*11:15*	*11:30*	*12:00*	*12:40*	*1:10*				Chewed gum, 11:20–12:45 p.m.
		0	6.9	7.2	7.2	7.2	6.9				
		1	6.2	6.6	6.5	6.5	6.3				
		0.5	5.7	6.2	6.2	6.3	6.0				
A. H.	7		*11:15*	*11:30*	*12:00*	*12:30*					Chewed gum, 11:16–12:01 p.m.
		0	6.6	6.9	7.2	6.7					
		1	5.7	6.3	6.5	5.7					
		0.5	4.9	5.3	6.0	4.9					
J. C. F.	8		*10:30*	*10:45*	*11:30*	*12:05*	*12:30*	*1:00*			Chewed gum, 10:37–12:40 p.m.; swallowed no saliva until 12:10 p.m.
		0	6.6	7.1	7.1	7.2	7.1	7.0			
		1	5.3	6.5	6.4	6.5	6.4	6.1			
		0.5	4.4	6.1	5.9	6.0	5.9	5.2			
J. C. F.	9		*10:55*	*11:30*	*12:30*	*12:55*	*1:55*				Chewed gum, 11:00–1:00 p.m.; sandwich and coffee, 1:20 p.m.
		0	6.7	6.9	7.0	6.9	6.3				
		1	5.7	5.7	6.1	6.2	5.0				
		0.5	4.8	4.1?	5.3	5.7	4.3				
J. C. F.	10		*12:00*	*12:08*	*12:20*	*12:30*	*12:40*	*1:00*			Chewed buttered toast, 12:01–12:05 p.m.; did not swallow it; repeated, 12:32–12:35 p.m.
		0	6.5	7.1	6.7	6.7	6.9	6.9			
		1	5.7	6.6	5.4	5.7	6.2	5.9			
		0.5	4.8	6.3	4.5	4.8	5.7	4.7			

When acid was added, it was always found advisable, after stirring the solution, to remove the electrode and wash it with distilled water

TABLE 4

Relationship between neutralizing action, phosphorus concentration and CO_2-combining power of saliva

SUBJECT	HCl ADDED cc.	COLLECTION PERIOD 1 pH	1 P, mg.	1 CO_2	2 pH	2 P, mg.	2 CO_2	3 pH	3 P, mg.	3 CO_2	REMARKS
C. P.	0	6.8	...	8.4	7.2	...	22.9	7.0	...	21.5	Gum chewed continuously after first sample collected
	1	5.9			6.7			6.6			
	0.5	5.0			6.2			6.3			
C. P.	0	6.8	18.7	9.0	7.2	12.8	18.8				Same as above
	1	5.9			6.4						
	0.5	5.1			5.8						
C. P.	0	6.7	18.9	7.5	7.0	16.9	14.2				Same as above
	1	5.9			6.4						
	0.5	5.0			5.7						
J. C. F.	0	6.5	19.8	8.9	7.0	16.0	12.5				Same as above
	1	5.7			6.0						
	0.5	4.9			5.1						
J. C. F.	0	6.7	18.6	10.4	7.0	14.0	15.5	6.9	14.2	18.5	Same as above
	1	5.7			6.1			6.2			
	0.5	4.8			5.3			5.7			
J. C. F.	0	6.8	17.1	13.1	6.9	14.0	13.5	7.2	15.6	18.8	Same as above
	1	6.0			6.0			6.3			
	0.5	5.1			5.2			5.8			
R. C. M.	0	7.1	16.4	23.1	7.2	15.0	25.9				Same as above
	1	6.5			6.6						
	0.5	6.2			6.3						
R. C. M.	0	7.1	...	18.0	7.2	...	25.0	7.2	...	23	Same as above
	1	6.4			6.9			6.9			
R. C. M.	0	6.9	15.2	14.4	7.1	12.2	22.9	7.1	13.9	22.9	Same as above
	1	6.1			6.6			6.6			
A. B.	0	7.1	13.5	14.5	7.2	11.0	13.5				Same as above
	1	6.2			6.2						
	0.5	5.3			5.2						
A. B.	0	6.8	...	8.0	7.1	...	10.9				Same as above
	1	5.4			6.2						
	0.5	4.7			5.2						
W. P.	0	7.0	14.0	19.3	7.2	12.6	21.3	6.8	15.2	14.7	Sample 3 collected after gum chewing stopped
	1	6.3			6.6			6.1			
	0.5	5.8			6.3			5.3			
Miscellaneous samples collected without gum chewing											
	0	7.1	14.2	18.5	6.8	16.0	14.5	6.6	19.4	8.9	
	1	6.2			6.1			5.7			
	0.5	5.7			5.2			5.1			
	0	6.2	...	4.1	6.9	18.0	10.7	6.7	17.7	8.8	
	1	5.2			5.7			5.2			
	0.5	4.5			4.9			4.6			
	0	7.0	...	23.0	6.8	11.3	4.1	6.9	10.5	10.0	
	1	6.2			5.2			5.7			
	0.5	5.8			4.2			4.8			

before finally saturating the solution with expired air and determining the pH. If this is not done, equilibrium is only slowly established, especially in mucinous saliva, possibly owing to the coating of the electrode by protein and thus hindering rapid development of a state of equilibrium between the electrode and the surrounding medium. Three cc. of saliva were used in all determinations, and exactly 1.5 cc. of $N/50$ HCl added in two portions—1 and 0.5 cc., respectively. The pH of the solution was determined after each addition.

Phosphorus was determined as follows: 1 cc. of saliva was added to 4 cc. of 10 percent trichloracetic acid in a 15 cc. centrifuge-tube, the solution thoroughly mixed, and centrifuged after a few minutes. Of the supernatant fluid, 2 cc. were transferred to a test tube graduated at the 10-cc. mark, about 5 cc. of water added, and phosphorus determined colorimetrically by Fiske and Subbarow's method for blood phosphorus. Occasionally a slight precipitate formed after adding the ammonium molybdate; when this happened, it was removed by centrifuging before comparing the solutions in a colorimeter.

CO_2-combining power was determined in the usual Van Slyke apparatus for blood analysis. Since the CO_2-combining power of the saliva is normally much less than that of blood, 2 cc. portions of saliva were allowed to flow as completely as possible into the evacuating chamber, one drop of caprylic alcohol added, and then sufficient normal sulfuric acid used to make the total volume of the mixture in the chamber 2.5 cc. A small amount of the saliva adheres to the walls of the glass and does not enter the evacuating chamber. There is a slight loss of carbon dioxide in transferring the saliva from the conditioning separatory funnel to the evacuating chamber. These unavoidable errors must, however, be quite small, and should not materially influence the results. The solubility of oxygen, nitrogen, and carbon dioxide in saliva is considered to be the same as in blood. The CO_2-combining power was calculated in the usual manner, excepting that, when the regular tables for blood analyses were used, the recorded value was divided by two, since 2 cc. of saliva were used instead of 1 cc. of blood.

Discussion. It will be seen, from *tables 1* and *2*, that the pH and the acid-neutralizing action of saliva rapidly decrease after a light lunch of sandwiches, etc.; also after a meal of cereal, fruit, toast and

coffee. Some decrease was obtained after a regular dinner of meat, vegetables, bread, dessert and coffee, but it was not so marked as in the other case. Oranges, when taken alone, did not cause any decrease in neutralizing action. Starr (5), and Henderson and Millet (6), previously showed a similar effect of meals on salivary pH. The slow rise in neutralizing action after meals makes it imperative, in comparative studies of salivary neutralizing action, to collect the saliva either before breakfast, or at a time at least three hours removed from the last meal.

No adequate explanation for the decrease in neutralizing action following a light meal seems available. It can hardly be a reaction to a

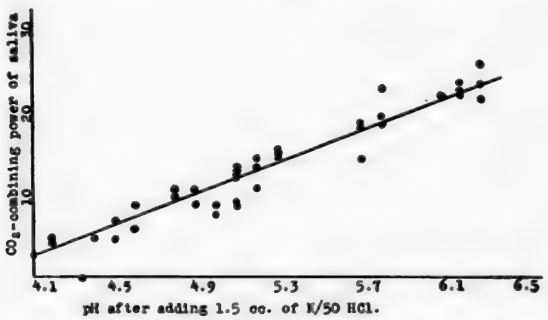

FIG. 2. GRAPH SHOWING RELATIONSHIP BETWEEN ACID-NEUTRALIZING ACTION AND CO_2-COMBINING POWER OF SALIVA

preceding increased neutralizing action produced by the mechanical stimulation of the food in mastication, since the very marked increase resulting from chewing paraffin or gum (*table 3*) is followed by a decrease to only approximately the normal value and not below it, as is the case when a light meal is taken. Similar results were obtained whether the period of chewing was long or short. It is possible that there is some relationship between salivary neutralizing-action and gastric hydrochloric-acid secretion. However, if this were the case, one would expect the meat extract to have a far more definite effect than that actually obtained. Further work is in progress to determine whether any relationship between the two does exist.

It will be seen, from the data in *table 4*, that in the same individual there is an apparent reciprocal relationship between acid-neutralizing

action and phosphorus concentration, but not a general relationship between the two. There is, however, quite a definite relation between acid-neutralizing action and CO_2-combining power. This can readily be seen in *table 4;* also in *fig. 2*, where a number of points are shown that are not given in *table 4*.

Summary

(1) The acid-neutralizing action of saliva markedly falls half an hour or so after a breakfast or light lunch; only a slight fall follows a regular dinner of meat, vegetables, bread, dessert and coffee. Oranges, when taken alone, caused no fall in neutralizing action.

(2) Paraffin- or gum-chewing markedly increases salivary neutralizing action; but it rapidly falls to the normal value after chewing is stopped. This indicates that the decrease *below* the normal value, after a light meal, is not due to the mastication process.

(3) In the same individual a reciprocal relationship seems to exist between salivary neutralizing-action and phosphorus-concentration, but there is no general relation between the two.

(4) There is an apparent relationship between neutralizing action and CO_2-combining power.

It is a pleasure to acknowledge the kindly help of Drs. W. B. Gurley and A. O. James, and other members of the Dental Infirmary Staff, in obtaining the data here presented. My thanks are also due the Chemical Foundation, Inc., for a research grant to our institution, which made possible the carrying out of this investigation. The meat extract (Valentine's Meat Juice) was kindly supplied by the Valentine Meat Juice Company, Richmond, Va.

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STUDIES OF THE CAUSE AND NATURE OF DENTAL CARIES[1, 2]

J. J. ENRIGHT,[3] PH.D., H. E. FRIESELL,[4] D.D.S., F.A.C.D., AND M. O. TRESCHER,[5] M.S.

Mellon Institute and School of Dentistry, University of Pittsburgh, Pa.

CONTENTS

I. Introduction 759
II. Analysis of literature and plan of attack 761
III. Evaluation of the objections to Miller's bacterio-chemical theory 776
IV. Studies of the effects of acids on enamel 786
V. Bacteriological survey of carious and non-carious patients 806
VI. Nomenclatorial study of lactobacilli associated with dental caries 813
VII. Summary and conclusions 824
VIII. References to literature 825
IX. *Discussion* 828
William J. Gies 828
Russell W. Bunting 831
E. H. Hatton 836
F. E. Rodriguez 838
Philip Jay 842
Theodor Rosebury 845
R. E. Morgan 850
Dr. Enright 850
Dr. Friesell 851

I. INTRODUCTION

The etiologic and preventive aspects of dental caries are the major problems that confront the dental profession today. The wide preva-

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[2] The Mellon Institute has paid the expense involved in adding this paper and discussion to this issue. If this expense had not been met in a special way, the paper could not have been published before a large accumulation of accepted manuscripts had been given precedence, which would have caused a delay of six to nine months. The grant from the Mellon Institute enables us, without expense to the *Journal*, to increase the size of this issue and volume by the number of pages in this paper and discussion.—[*Ed.*].

[3] Senior Fellow, Mellon Institute.

[4] Dean, School of Dentistry.

[5] Formerly Junior Fellow, Mellon Institute.

lence of dental caries, with its consequent causation of pain, of interference with proper mastication and digestion, and of loss of esthetic values, has convinced dentists generally of the importance of the unsolved problems connected with the disease. In recent years two new developments have made more urgent than ever the need for intensive research to solve these problems. These two developments have been (*a*) the demonstration that dental caries is often an important link in a chain of more serious illnesses, and (*b*) the growing disagreement with generally accepted views on the etiology and prevention of tooth decay. It is not within the scope of this paper to discuss the relationship of tooth decay to the more serious diseases with which it has been associated, or for which it may be a causative factor. It is sufficient to point out that, despite the paucity of accurate knowledge on many questions concerning the pulpless tooth and the dental zone as foci of infection, ample evidence indicates that a diminution in tooth decay would be accompanied by a drop in the incidence of arthritic, cardiac, and renal diseases. We are especially concerned in this contribution with the second new development—that of the marked difference of opinion on the nature of the etiologic factors in dental caries.

During the past decade there has been a large and increasing group of dentists who believe that local environmental factors play no part or only a minor rôle in the causation of tooth decay. This school of thought maintains that any program of prophylaxis based on oral hygiene is bound to be of slight value, and that our only hope of combating caries successfully is through proper diet. When a specialist as prominent in the dental profession as Howe voices the doctrines of this group, it is the occasion for considerable concern among dental authorities who are responsible for the widespread oral-hygiene movement among federal, state, and municipal agencies.

The clearly pressing need for knowledge relative to the pertinent factors that are concerned with the cause and prevention of dental caries brought about, in 1923, the foundation of a Multiple Fellowship at Mellon Institute. The studies of this Fellowship have been continuous for the past nine years, during which the coöperation of many specialists in the University of Pittsburgh School of Dentistry has maintained an enthusiastic and broad outlook on the problems under investigation. During the first two years, Friesell and Vogt (20) made

an extensive study of the relation of the pH, and of the calcium and phosphorus contents, of saliva to the occurrence of dental caries; also of the effects of acids on enamel. The results of these investigations led to the conclusion that no correct solution of such a complex problem as the cause and nature of dental caries would be possible without focussing thereon the combined resources of modern scientific knowledge in the fields of dentistry, histology, chemistry, bacteriology, and nutrition. The following report deals particularly with the studies of the last six years and is the first complete presentation of our findings up to this time.[6]

It is a pleasure to acknowledge the collaborative aid of the scientific staffs of the University of Pittsburgh School of Dentistry, and of the Mellon Institute. Drs. W. F. Swanson and H. D. Cossett have given valuable help in the histological research; Drs. C. W. Hagan and T. J. McBride in painstaking clinical inquiry; and Dr. J. C. Esselman in the x-ray field. The authors are particularly indebted to Mr. F. W. Nitardy, the Donor's representative, and to the staff of Mellon Institute, especially Drs. George D. Beal and William A. Hamor, for their continuously active interest and constructive aid during the entire investigation.

II. ANALYSIS OF LITERATURE AND PLAN OF ATTACK

Although a thorough examination of the literature on dental caries revealed that the etiologic factors are quite complex, involving obscure influences of genetic and endocrine character, it was concluded that a broad study of the cause and nature of dental caries really resolved itself into a search for the strong points and shortcomings in the statements of the members of two schools of thought, which are not always clearly defined. One of these schools maintains that local environmental factors are chiefly responsible for caries; the other school holds

[6] "Studies of the Effects of Acids on Enamel" was presented at the regular monthly meeting of the Pittsburgh Section of the International Association for Dental Research, School of Dentistry, University of Pittsburgh, March 3, 1930; "The Bacteriological Survey of Carious and Non-Carious Patients," at the regular monthly meeting, March 7, 1932; "The Nomenclatorial Study of Lactobacilli Associated with Dental Caries," at the meeting on April 3, 1932. The first complete presentation of this entire report was made at a special meeting of the Pittsburgh Section on June 23, 1932; *Journal of Dental Research* 1932, xii, p. 569; Aug.

that deficient diet and defective nutrition are primarily and sufficiently responsible for the causation of decay. After attempting to decide what was true and what were the gaps in the knowledge in these two schools, our plan of study was designed to fill in the missing information and to extend the boundaries of knowledge in that school which is concerned with the active rather than the predisposing factors in the causation of decay of enamel.

A. Status of knowledge in the local environmental school

As the school that emphasizes the importance of local environmental causes is the older, we shall first consider briefly the status of knowledge within that group. Miller (60, 61) did an excellent piece of work as a pioneer in demonstrating that mouth organisms produce lactic acid in fermenting carbohydrate-food debris, and that lactic acid decalcifies tooth enamel. He also pointed out that the decalcification of enamel in the mouth occurs at the "retention centers," pits and fissures, and proximal surfaces. The fact that Miller's conception gave no consideration to several characteristics of dental caries that dentists have generally observed explains why it is referred to as "Miller's *theory*." Very prominent among the shortcomings in Miller's results was the absence of explanations as to why mouths in filthy condition are often free from caries and also why natural carious processes often come to a halt. It is well known that Miller was unable to give very accurate or complete information of a bacteriological nature, because the science of bacteriology was only in its infancy when he carried on his studies. In addition to Miller, Black (2), Williams (81, 82), and Gies and his collaborators (21, 22, 23, 24, 25) have made outstanding contributions to the knowledge of local environmental influences on dental caries. More recently McIntosh (57), Rodriguez (71), and Bunting (8, 9, 10, 11, 12) have given more detailed bacteriological information of the carious process. These latter three investigators agree as to the importance of lactobacilli in producing the acid that decalcifies enamel.

But after all this research, there still remain several gaps that were pointed out after Miller's classical contributions. In consequence, the last decade has brought into ascendancy the group, led by Howe, who maintain the primary importance of proper diet. The gaps that have

been recognized from time to time, in the view that dental caries is caused by the fermentation of food debris on the teeth, may be summarized briefly as follows:

1. The carious areas produced artificially by acid have not simulated natural caries in certain particulars; namely, invasion of the enamel to the dento-enamel junction, with surface continuity still intact accompanied by typical flame-like spread of the process at the dento-enamel junction.

2. There has not been a sound explanation of the halt that occurs in carious processes for indefinite periods.

3. Proponents of the importance of local environmental causes have not explained satisfactorily why mouths in filthy condition are frequently free from caries.

4. The local environmental school has not set forth plainly why caries is more prevalent, and makes more rapid progress, during pregnancy and in certain constitutional diseases.

5. Caries has not been produced experimentally in animals by feeding diets containing large amounts of sugars and starches, even where the carbohydrates adhered to the teeth and caused an overgrowth of aciduric microörganisms.

It is possible that two of these gaps, numbers (4) and (5), are merely fancied deficiencies in evidence. But even though only three of the statements could be used justly as arguments against the soundness of Miller's conception, especially after its forty years of acceptance, there seems ample justification for further search for a more complete explanation of the carious process. This question becomes more pertinent when one considers the lack of effectiveness in reducing the incidence of caries by an oral prophylactic program based on the views of Miller, Black, and Williams relative to the important factors in the etiology of caries.

B. Status of information within the nutritional school

Within the dental field, it appears that Mellanby (58, 59) and Howe (44, 45, 46) have made the most extensive contributions on the relation of deficient diet and defective nutrition to the etiology of dental caries. Since 1918 Mrs. Mellanby has published valuable reports of studies on the relation of diet to dental caries. She has found that, by

feeding diets deficient in vitamin D to dogs while their teeth are in the developmental period, the teeth when erupted are defective in structure and improperly placed in poorly calcified jaws. She has also ascertained that certain cereals in the diet, notably oats, cause similar effects, if a superabundance of vitamin D is lacking in such a diet. She and her associates have reported like results in similar experiments on rabbits, rats, and man. Other macroscopic and microscopic studies by Mrs. Mellanby of the tooth enamel of children have given evidence that leads her to associate the presence of dental caries with defective tooth structure. There is doubt whether the teeth that were at her disposal for such a study have provided a satisfactory foundation for such a correlation. Although the conclusion that she has offered may be true, the fact that the teeth studied were chosen at random from extracted and exfoliated specimens, and did not include the teeth that remained in the mouths of the same individuals, gives a foundation for questioning the justification for her claims. According to Mrs. Mellanby, the type of diet with a sufficiency of vitamin D that causes the development of sound teeth properly placed also tends to prevent dental caries in teeth that have been erupted. She speculates that this effect may be due to increasing the resistance of the teeth. She alludes to the possibility of variations in the saliva giving the increased resistance, but does not place much emphasis on this factor. She states that the bacteriological factor in the causation of dental caries is relatively unimportant when the teeth are well formed, and that it is significant only when teeth are defective in structure or improperly placed in the jaws.

Howe (44, 45, 46), in his nutritional studies of dental caries, used the guinea pig in preference to the dog or rat, and has come to the conclusion that dietary deficiency in vitamin C is the important cause of the prevalence of dental caries. He has found that, whereas the normal odontoblastic layer of guinea pigs consists of cells in orderly array and closely applied to a homogeneously staining dentin, scorbutus is evidenced earliest (7–12 days) by the separation of this layer from the dentin by a narrow margin, occasional irregular calcium deposits in the odontogenic zone, and irregularities in the odontoblastic layer. After a longer time (12–14 days), there is complete separation of odontoblasts from dentin. There was rupture of the processes of odonto-

blasts; and the spaces separating odontoblasts and dentin contained no stainable material. The odontoblasts showed changes, the chief of which were diminution in size and increased density of staining. There were also changes in the pulp. Finally, in absolute scorbutus, he found a shrunken pulp completely freed from the dentin and apparently floating in a liquid material. The new formation of intracellular material of the matrix of bone and of dentin had ceased. In twenty-four hours, the administration of only 2 cc. of orange juice to a guinea pig kept for twelve days on a scorbutic diet resulted in the formation of a zone of dentin on the separated odontoblast. In three days, 8 cc. of orange juice given daily resulted in the complete filling of the space between the dentin and odontoblasts in scorbutus of long standing. Howe (45) has also carried out an extensive investigation on monkeys. By withholding vitamin C and producing scorbutic animals, he has observed changes in the supporting structures of the teeth, both in the hard and soft tissues. He has also reported, in addition to the mandible he extensively decalcified, caries in the first permanent molars and at the gingival margin of the lateral and central incisors. In other experiments some caries has been found even when the daily dose of orange juice was as high as 26 to 30 cc. In these as well as other experiments Howe observed that, with diets deficient in calcium and phosphorus, and in the amounts of antiscorbutic factor, protein, and indigestible residue, the teeth were particularly susceptible to dental caries and other dental disorders.

Hanke (32, 33) agrees with Howe as to the importance of vitamin C in the diet to prevent dental caries. From a statistical study of 191 patients under dietetic treatment and observation, he saw improvement in the dental conditions of 104 patients. He observed that all the patients susceptible to dental caries were on a diet low in vitamin C. Of 104 patients who were fed one to two pints of milk, a moderate amount of meat, fresh vegetables, from a quarter to one head of lettuce, the juice of one-half a lemon, one pint of orange juice, and from one to two eggs daily over a period of eight months, there were 60 in whom no new cavities developed during the period of observation, but in whom caries had been previously reported to be active.

Grieves (28) examined the teeth of hundreds of rats in McCollum's laboratory, and learned that deficiency of vitamins A and D, and of

calcium and phosphorus in improper ratios, caused the development of poor teeth.

Boyd and Drain (6, 7), in studies of children fed milk, fresh fruit, vegetables, and cod-liver oil, observed that open cavities of teeth did not increase in size, and that the walls of the cavities became dark and hard in character.

Hawkins and his associates (35, 36, 37), in interesting contributions to the subject of the relation of nutrition to dental caries, have emphasized the importance of diets designed to yield a high alkaline ash and an increase in calcium to enable the saliva to neutralize the acids of fermentation. After obtaining a lead as to the importance of certain nutritional factors in the causation of dental caries, Hawkins made a large series of salivary analyses, the results of which apparently strengthened his opinion as to the importance of the high alkaline ash and calcium content of saliva. He rounded out his investigation by a clinical study, in which 74 children in an orphanage were on diets containing moderate amounts of meat and cereals and having a high alkaline ash and calcium content. Those children whose saliva was low in calcium and alkalis were fed cod-liver oil and orange juice. He reports that no new cavities were found, but that there was a marked increase in the deposit of calculus.

Höjer (39) demonstrated that the odontoblasts are peculiarly susceptible to injury through deficiency of vitamin C. He showed that, when guinea pigs are fed for from twelve to fourteen days a diet containing about 80 percent of the minimum protective dose of the antiscorbutic vitamin, the cells constituting the odontoblastic membrane become uneven in length and present a somewhat serrated margin. When about half of the minimum protective dose is fed, the odontoblastic membrane separates from the dentin, apparently with a detachment of the fibers that penetrate the dentinal tubules and pass to the base of the enamel. When 30 percent of the minimum protective dose of vitamin C is fed, the odontoblastic layer breaks up into islands of distorted cells. No one has studied the extent to which the odontoblasts may return to normal after a specific amount of injury has been effected by this procedure.

Bunting (13), who has strongly advocated the importance of local environmental factors, seems to have been converted to the importance

of proper diet in combating caries. In an extensive clinical study, in which he used dietary measures and hexylresorcinol solution (S.T. 37) diluted with three parts of water, he concluded that, of the two methods employed, diet was by far the more active inhibitive force.

There is also a wealth of information on the relation of diet to dental caries in the investigations of Hrdlicka (48), Waugh (78, 79, 80) and Leigh (52), who have studied the conditions of the teeth of races living on diets of a distinctive character. These investigators agree that certain races that had proteins as practically their only food suffered little from dental caries. McCollum (56) reports that Hrdlicka says that northwest Greenlanders have the best teeth of any fairly large group of people in the world today. These people exist essentially upon a carnivorous diet, consisting of eggs and the flesh of birds, the beluga whale, and the norwal. Leigh examined 324 Eskimo skulls and confirmed the commonly held opinion that these people have very little caries. Waugh made a study of the resultant effect on the teeth of the changing diet of the American Eskimo. Both in Labrador and Alaska he found that the old Eskimos, who in childhood had lived on the primitive native foods of their ancestors, even though small in stature, had very large, well formed and regular jaws, and decay of the teeth was almost absent. From studies made in Labrador, Waugh believed that the incidence of caries was in direct proportion to the amount of carbohydrate in the diet.

The information that has been accumulated on the relation of diet to dental caries is convincing testimony on the fact that improper diet is at least an important predisposing factor in the causation of tooth decay. It is apparent, however, that our knowledge on this subject is still quite meagre and debatable: Howe and Hanke emphasize the importance of vitamin C, and disagree with Mellanby, who holds that vitamin D is the important factor; while Hawkins and others lay particular stress on the importance of diets that give a high alkaline-ash yield and a high calcium content in the saliva. More recently, two other contentions have been emphasized, with Hoppert *et al.* (42) finding that the physical character of the diet as regards its retention by the teeth is the important factor, while Klein (50) stresses the importance of the phosphorus level in the diet and saliva.

Although considerable constructive knowledge of the cause and

nature of dental caries has come from these nutritional studies, much confusion in the minds of dentists has arisen from the interpretations of certain investigators who, in attempting to emphasize the value of nutritional factors, seem to discount the importance of the effects of the local acids of fermentation in causing decay of the enamel. Bunting, Drain, and Bödecker, who have reported evidence that has been used to strengthen the claims of the nutritional school, have not abandoned the position that attributes direct active importance to local environmental factors.

It is difficult to learn from Howe's reports generally whether he has abandoned belief in the importance of local environmental factors. In 1921, however, he made this statement: "If Miller's theory is sound, it should be an easy matter to produce tooth decay in animals by fermentation. We have fed guinea pigs upon diets containing large amounts of sugars and starches. This diet was continued for from six months to a year. The sugars were readily eaten and adhered constantly to tooth surfaces. The flora of the mouth became aciduric in character, but no effect could be detected in any of the teeth." From this expressed view one may believe that, at that time, Howe held the opinion that Miller's theory was not sound. It appears that Howe did not consider when he made the above statement that the opinion of bacteriologists had markedly changed since the time when they accepted Koch's postulate as to the need for reproducing a disease in experimental animals with a culture of the suspected etiologic agent. He must have learned of this change between 1921 and 1926, because at the later date he took the more recent bacteriological view when he reported (46): "While the ineffectiveness of carbohydrate fermentation acting locally in the production of caries in experimental animals may not necessarily confute Miller's theory, it does apparently relegate it to a secondary position." A study of Howe's reports since 1921, and even a conversation with him on the subject, have failed to inform the authors whether he has definitely abandoned Miller's ideas on the importance of the local environmental agencies in actively causing caries. It is known, however, that an appreciable number in the dental profession and several investigators are interpreting Howe's reports to mean that he claims that the active cause of dental caries is metabolic change in the enamel due to deficient diet and defective nutrition.

Relative to this, Williams (82), whose opinion on dental caries must always be given careful consideration, remarks: "I freely accept the evidence he presents, as to facts, of the results of all his valuable work but the evidence presented does not warrant some of his conclusions. If metabolic change in enamel were the chief cause of caries, then cavities might form internally or at any part of the tooth and they would be just as likely to form on the labial surface of a tooth as elsewhere. But we all know such is not the case."

Consideration of two undisputed facts—(a) caries of the enamel begins at the outer surface, and (b) caries occurs in the pits and fissures and proximal and gingival areas—places one in a strategic position to interpret the results of Howe's experiments in which he used diets deficient in minerals and vitamin C. There appears to be doubt of the importance of the two latter factors in actively causing caries of the enamel, especially if there is no vital circulation or no metabolic activity in fully erupted enamel. Although leading dental histologists, such as Hopewell-Smith (41), Williams (82), Noyes (66), and Chase (14), have demonstrated satisfactorily to an overwhelming majority of dentists that no such circulation occurs in enamel, Bödecker (5) and Fish (17, 18) take issue with them. A study of the reports of Fish indicates that he appears to have demonstrated a circulation in dentin, but has presented no evidence to justify a similar conclusion with respect to enamel. Concerning the enamel, Bödecker (3, 4) has made a contribution to our knowledge of dental histology in demonstrating the presence of organic sheaths around enamel rods; but his claims that these sheaths are nutrient channels in the enamel have not been substantiated, Rosebury (73) having shown that these sheaths consist mainly if not wholly of keratin.

The results of an experiment carried out by Gies (26) for a period of nearly ten years should convince us that there is no vital circulation in enamel. Gies repeatedly injected trypan blue into the abdominal cavity of young dogs at short intervals, beginning on one shortly after birth and continuing with the injections occasionally while the teeth were in process of formation. All the tissues were deeply stained. As the teeth erupted, it was seen that all the enamel was likewise deeply stained. In other dogs of the same litter, similarly treated during successively later periods of enamel formation, the enamel which had

been completed before the first injection remained unstained. The dye rapidly disappeared from all the tissues except the enamel of the teeth (slowest from the bones and dentin), and after nine years (1917–26) it remained in the enamel without change. Williams reported this type of experiment with other animals, always with similar results. We confirmed these results of Gies and Williams by experimental observations on the enamel of the molars of rats.

Williams has also pointed out that production or repair of enamel after it has fully erupted could not be expected, because the ameloblasts, the only cells that possess the power of enamel formation, are lost at the time of eruption. There is of course the possibility of repair of decayed areas of enamel through the agency of the dentin, or by precipitation of salts from the saliva, but these are not the types of repair which seem to be meant by Bödecker. To an unprejudiced observer the fact that defective enamel, either carious or hypoplastic, does not repair itself demonstrates the lack of soundness in Bödecker's views on the histology of the enamel, or in Howe's views on the etiology of caries of enamel.

No statements have been seen in Hanke's reports that should cause one to discredit Miller. We agree with Hanke when he writes that "Miller's theory is far too simple to explain dental caries," and also when he observes that, "in spite of the fact that a campaign of cleanliness, alkalinization and gum massage has been waged for many years, caries and other dental disorders have continued." On the other hand, we disagree with Hanke with respect to the accuracy or pertinency of his statement that "a steady flow of saliva will rather effectively prevent an accumulation of acid anywhere in the mouth, and tricalcium phosphate, the chief constituent of the teeth, is not readily soluble in very dilute acid." The reasons for our disagreement will become manifest in the section of this report that deals with the effects of acids on tooth enamel. Hanke's question, "Why then do not the teeth in pyorrhea patients decay more rapidly in the presence of this increased number of bacteria?," is more relevant. He mentions these bacteria as being largely streptococci and spirochetes of an acid-producing type. It might be answered that none of the investigators, including Kligler, Rodriguez, McIntosh, Bunting and Jay, who have contributed to the weightier type of bacteriological reports

on the subject, has found that streptococci or spirochetes could be associated with the etiology of caries of enamel. But we must add that we have seen no evidence in any of their reports showing that acid-producing bacteria other than lactobacilli in the mouth are not involved in the decalcification of enamel. In the correlation of our study of the effects of acids on enamel with the results of our bacteriological investigation, there will be given evidence indicating that spirochetes or streptococci are not involved in the decalcification of enamel, because these organisms do not tolerate an acidity that is strong enough to decalcify enamel in the mouth.

It appears to us that the most liberal interpretation that at this time is justified by the progress of studies of the relation of nutrition to dental caries is this: (*a*) diets rich in vitamins A, C, and D, and yielding an alkaline ash, when utilized systematically during the developmental period of teeth, produce teeth less liable to decay; (*b*) continuation of a similar diet after the eruption of teeth causes the production of saliva having slight protective power against caries; and (*c*) deficient diet is important only as a predisposing cause of dental caries. At this point it seems pertinent to call attention to some of the outstanding clinical features of dental caries, which strangely enough appear to have been entirely overlooked by several other workers in this field of research.

C. Some clinical aspects of dental caries[7]

Dental caries is a chemical dissolution of the calcium salts of the enamel and dentin of the teeth. When caries attacks the enamel, the entire carious area is dissolved, because there is so little organic matter in it that it can not maintain its form, and a cavity results. When caries reaches the dentin the calcium salts are dissolved, but the matrix of organic material in dentin, while softened by the loss of the calcific material, is sufficient to retain the bulk and form of the dentin for some time. This organic matrix of the dentin is eventually decomposed and dissolved, and a cavity occurs in the dentin also.

The first sign of the beginning of caries on the enamel surface is an unnatural opaqueness caused by interference with the refraction of

[7] In this section one of us (H. E. F.) presents, as concisely as possible, the results of his practical and clinical observations since 1895, and also the results of his critical studies of the literature of the pathology of dental caries during this 37-year period.

light rays, owing to the irregular dissolution of the calcium salts of the enamel. The first appearance of caries in dentin is a softening of that tissue. Caries always begins on the surface of the tooth, never in the interior. The enamel must be decalcified before the dentin is reached; or, in those cases where dentin seems to be decaying much more extensively than the enamel surface, the causative agents have reached the dentin through defects or faults in deep pits or fissures that extend through the body of the enamel. These defects occur during the formation of the enamel, leaving microscopic openings from the mouth to the dentin where the enamel lobes have failed to join.

When caries attacks a tooth it does not affect the tooth uniformly all over its exposed surface, but begins at points so definite on the various anatomical surfaces of the tooth that an experienced operator can readily predict where decay will begin on a given surface, and to what limits it will extend on that surface. Some areas of tooth surface are practically never attacked by this disease. There are three general regions on tooth surfaces where caries begins; namely, first, in pits and fissures; second, on buccal and labial surfaces near the gum line; third, on proximal surfaces close to the contact point on the gingival side. Caries never begins on the occlusal surfaces of the teeth except in pits and fissures that extend through the enamel into the dentin as a result of faulty formation of the enamel, or in those rare cases where the pits or grooves are abnormally deep and narrow.

When caries begins in pits and fissures, it never spreads on the adjacent surface of the enamel, but develops laterally along the junction of the enamel and dentin, at the same time penetrating the tubular openings in the dentin toward the central portion of the tooth. In time, caries of this type will destroy a large part of the dentin beneath the occlusal enamel, and weaken it by decalcifying the inner surface of the enamel itself. Sometimes a large amount of the dentin forming the crown of the tooth will be dissolved, and the original opening through the enamel will not have enlarged. The patient may not be aware of decay in that tooth until the weakened enamel gives way under stress of mastication, crushes in—and the tooth is found to be "hollow." If caries is discovered early in pits and fissures, and the decayed tissue is removed and the cavity properly filled, no recurrence of decay around the margins of that filling will follow and there will be no further decay

on that surface of the tooth. Also, if such pits and fissures are ground out and made into smooth grooves with no overhanging walls, so that the food in mastication will sweep thoroughly over all portions of the exposed dentin, there will be no further decay. It is true that the dentin will wear away more quickly than enamel, but there will be no decalcification of dentin or enamel on the surface that is scoured by the food in the process of mastication.

Caries does not begin on the enamel of the buccal and labial surfaces where that enamel comes vigorously in contact with food during mastication, unless there is a fault or defect in the enamel surface, a rare occurrence on the buccal and labial surfaces of the teeth. When caries begins on these surfaces it is on the unscoured portion thereof, close to the gum line and about the center of the unscoured area. In this region caries spreads superficially toward the gum line, but never goes beneath it; it spreads toward the occlusal till it reaches an area where the scouring of the tooth prevents its further progress; mesially and distally it spreads nearly to the axial angles of the tooth crown, or to a point where the masses of food that sweep through the buccal or labial embrasures keep that surface scoured and polished. Caries that begins in pits and fissures does not spread on the surface, but penetrates toward the interior of the tooth. On the unscoured portions of the buccal and labial surfaces, however, caries spreads widely over the unscoured area and penetrates the enamel very slowly. We rarely find faults in the enamel on these surfaces. If fillings are made early in cavities of this kind and include all the injured area, but do not reach the gum line or scoured areas near the angles of the tooth, decay will continue around the best type of filling. Such decay recurs until the cavity outlines are extended to a point where the margins of the filling will be exposed to friction by the food. Fillings on these surfaces must have their outlines extended slightly beneath the gum tissue gingivally, to the axial angles mesially, distally and occlusally to a point where the food will keep the margins of the filling clean. When fillings are thus extended on these surfaces, decay stops and does not recur.

Caries does not begin upon a proximal surface until the septal gum-tissue between the teeth recedes from beneath the contact points and leaves exposed an area of proximal tooth surface that is not scoured by the grinding or sweeping of the food. Such cavities never extend

occlusally beyond the height of lateral contour on the proximal surface of the tooth, and their extension buccally and lingually is limited by the friction of the food as it sweeps through the buccal and lingual embrasures. In the case of proximal-surface caries, if the approximating tooth should be removed before the cavity has penetrated the enamel and involved the dentin, such decay usually becomes inhibited because the food sweeping through the space left by the missing tooth cleans that proximal surface and prevents further destruction of the enamel.

In deciduous teeth it is a common experience to find incipient proximal decay on the incisors and cuspid teeth becoming inhibited as these teeth separate because of expansion of the growing jaw, thus creating between the teeth spaces which permit food to pass and to keep those surfaces scoured. In mouths where the teeth have natural spaces between them and are not in close contact with each other, as is more commonly the case, proximal decay is rare. This can only be due to the scouring effect of food through those spaces, keeping the proximal surfaces clean.

Dental caries almost never occurs on the lingual surfaces of the teeth except in those cases of abnormal tooth formation where pits and fissures (owing to developmental faults) are present, as occasionally occurs in the upper central and lateral incisors. In neglected mouths, where cavities are progressing on the other surfaces of the teeth, these cavities are rarely found on the lingual surfaces because of the frequent and intimate contact of these surfaces with the food and tongue during mastication.

Clinical experience clearly demonstrates that there is nothing in the structure (as distinct from conformation) of the teeth that prevents caries. The localization of caries in its beginnings is clearly due to the fact that these susceptible areas are least affected by the friction of food. Poorly formed tooth structure, caused by malnutrition during its formative period, is not more subject to attack by caries than is sound tooth structure, unless it presents protected areas or spots not scoured by food. Hypoplastic teeth are frequently unaffected by caries when the hypoplastic areas are limited to portions of the tooth surface that are readily scoured in mastication.

Decay seldom begins on the axial angles of a tooth, when that tooth

occupies its normal position in the arch; but where a tooth is found out of position, and the axial angle is so placed as to be no longer exposed to friction, then cavities readily occur on the angle. In cases where the teeth have a quarter-turn in the arch, and the normal lingual surface of the tooth is in contact with an approximating tooth, caries readily begins on that lingual surface.

In cases of irregularities of the teeth, where one incisor sometimes overlaps its neighbor and caries begins on the proximal surface, the carious area follows the surface lapped by the approximating tooth instead of taking the ordinary form of proximal decay in a tooth in its normal position. If a clasp or appliance is closely fitted to a tooth and improperly cemented at a given spot, fairly rapid caries can be induced in a mouth that is otherwise free from decay. It is common clinical experience to find decay rapidly taking place under a leaky filling, because of poor adaptation of the filling material to the cavity walls, when decay is not in progress elsewhere in the mouth.

It is a well established fact that in former days, when it was customary occasionally to use natural teeth in transplantation operations—or the crowns of natural teeth, human or animal, for attachment to plate bases—such teeth decayed frequently in the mouths of the wearers; and where decay occurred, it simulated decay in natural human teeth in every respect. The fact that caries never occurs or progresses under a deposit of tartar on the teeth accords with the general observation that caries always begins on the outside of the tooth and progresses inward; never on the inside, outward.

Caries is a slow process and may progress microscopically for a considerable period of time before definite injury of the tooth structure can be recognized. On those surfaces of the tooth that are frequently scoured and polished by the friction of food, decays do not occur. This clinical observation has given rise to the statement that if a tooth is kept clean it will not decay. It does not decay on those surfaces that are kept clean. If the areas of tooth structure that are most susceptible to the beginnings of decay are cleaned with sufficient frequency, decay will not occur on those areas. Skillful, conscientious operators who recognize the areas of tooth surfaces that are susceptible to attack from caries and who, in preparing cavities, include all such areas in the cavity outline, obtain clinical results that bear ample sup-

port for the conclusions that dental caries always begins on the outer surface of a tooth, and that when the surface is properly protected the tooth will be preserved against dental caries.

D. Plan of experimental attack

This consideration of the merits of the views of the nutritional school and of the contentions of the local-environmental group has brought us to the conclusion that Miller's views, while not sufficiently far-reaching, can be more logically correlated with the *clinical* disease; whereas the defects in Howe's conception are such as to make a correlation impossible. Therefore, the following plan of study was projected: (*a*) Consider the seriousness of the objections to Miller's theory, and determine whether any or all of these objections can be removed. (*b*) If Miller's main conception should be substantiated, conduct a comprehensive study of the effects of acids on enamel, the need for which cannot be overemphasized. (*c*) Study the effects of variations in saliva on the progress of dental caries. (*d*) Make a bacteriological survey of carious enamel processes.

III. EVALUATION OF THE OBJECTIONS TO MILLER'S BACTERIO-CHEMICAL THEORY[8]

Objection 1. Artificial acid-decalcification of enamel does not simulate natural caries in allowing a surface continuity to exist concurrently with evidence of the carious process spreading at the dento-enamel junction directly under the intact carious enamel.

This was an objection to the acceptance of Bunting's (8) contention that caries resulted from an acid decalcification in which Bacillus acidophilus was the chief etiological factor. Photomicrographs of artificial caries that Bunting presented led Beust to say: "I feel that there has been no relation shown between B. acidophilus and caries, for the simple reason that teeth do not decay in the manner shown on the screen. In incipient caries in the mouth, we have advancement of decay through the enamel by degrees; at first, a small area, followed by gradual penetration, until finally the dentin is reached. In Dr. Bunting's presentation, we have seen layer after layer of the tissue removed. In caries in the mouth, we find that the tooth does not decay in this

[8] These objections were summarized on page 763.

manner, for here the acid in its advancement toward the dentin leaves the enamel intact. The form of the enamel rods, the matrix, if you will, remains complete until almost all of the enamel has been traversed by the agencies that are attacking the tooth." Beust's point appears to be well taken in a percentage of cases in the light of our study of ground thin sections of natural carious lesons. It appears that Bunting's artificial lesions did not closely approach the appearance of natural carious lesions because his acid solution was too strong or the immersion period was too long, or both.

In our efforts to obtain artificial caries that simulated natural lesions we exposed small areas on the labial surface of extracted central incisors, through windows in asphaltum coverings, to a wide range of acid concentrations of both lactic and citric acids for different periods of time extending up to sixty days. It was relatively easy to obtain artificial lesions that quite closely simulated natural incipient caries of the enamel both macro- and microscopically (*fig. 1*). The best section of a more advanced artificial lesion is shown in *fig. 2*. This was produced by allowing a lactic acid solution (pH 4.6) to act on the enamel for fourteen days. Although this section falls far short of having surface continuity, the fact that an appreciable amount of intact enamel-matrix remains over the area where there is a typical natural spreading of the lesion at the dento-enamel junction is a distinct advance, particularly in the light of the following considerations. Natural carious lesions of enamel in the mouth occur on tooth areas that possess a thinner layer of enamel (*fig. 3*) than that of the labial tooth surfaces to which our experiments were confined. If the enamel on the tooth where we direct the action of lactic acid (pH 4.6) for fourteen days had been no thicker originally than the resultant layer of intact enamel-matrix over the damaged dentin, we might have satisfied the desire for both surface continuity and injured dentin in the same ground tooth-section of artificial caries. *Fig. 4* is presented to show that considerable penetration of enamel by acid is possible without loss of surface continuity.

But one might ask: "Why not produce the artificial lesions at the gingival margins, for instance, where the enamel is relatively thin?" The answer is that the use of these areas in our experiment is precluded by the probability that natural carious lesions are already present at

these areas. The only desirable teeth available to us for experimentation are those extracted from human mouths. In such a group of teeth

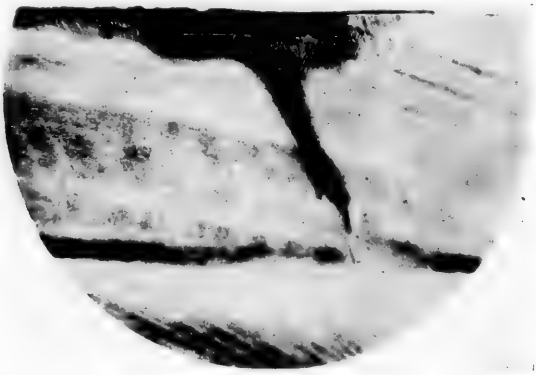


FIG. 1. Artificial caries that simulates incipient natural caries. Produced by lactic acid (pH 5.0) in 24 hours. ×40

FIG. 2. More advanced artificial caries, with beginning spread of process at dento-enamel junction typical of natural caries. Produced by lactic acid (pH 4.6) in 14 days. ×40.

the percentage having carious lesions at the general retention centers is very high, as is evidenced by microscopical study of ground sections. There is no doubt that the most expert dental examiner, with the aid

FIG. 3. Natural carious lesion at gingival third, with typical natural spread at dento-enamel junction. ×40.

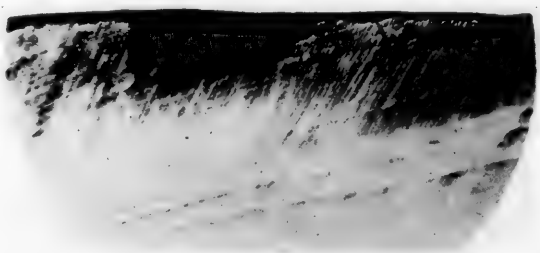


FIG. 4. Artificial caries. Fair amount of penetration. with surface continuity. ×40.

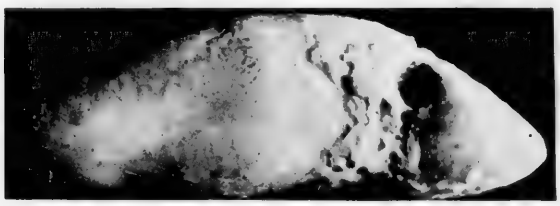
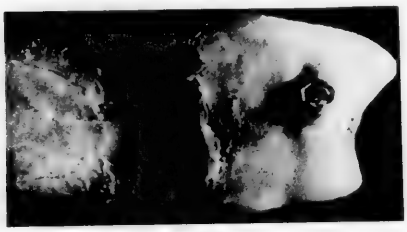


FIG. 5. Insoluble area that resulted when entire tooth was immersed in *N* lactic acid for 12 hours.

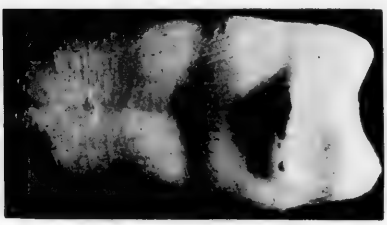
of all facilities at his disposal for detecting *incipient* caries of the enamel, would miss many areas that would show such caries on microscopical examination of ground sections.

This subject of the comparison of natural and artificial lesions could be discussed constructively at much greater length, but sufficient information has been presented, we believe, to convince a student of the subject that artificial carious lesions of the enamel, prepared by the action of lactic acid, can be produced to resemble natural caries when examined in section.

Objection 2. There is no experimental evidence to explain why the natural carious process often comes to a halt.



FIGS. 6 AND 7. Two views of acid-insoluble wall around cavity. This wall took above indicated form when entire tooth was immersed in 5 *N* nitric acid for 10 minutes.



FIGS. 8 AND 9. Two views of acid-insoluble proximal caries of enamel. This prominence resulted when entire tooth was immersed in 5 *N* nitric acid for 10 minutes.

Kligler and other investigators have called attention to this defect in Miller's theory. The troublesome feature about this observation is that aciduric organisms are present in such carious lesions. Hadley (29) suggests the existence of a symbiotic relation of the aciduric bacteria with a proteolytic type that forms a product capable of neutralizing the acids of fermentation. It seems likely that some of the observed interruptions of carious processes have been in cases that involved the dentin. The evidence from the studies of Mellanby, Boyd

and Drain, and other nutritionists indicates such a halt was caused to some extent by the production of secondary dentin in a desirable reaction by the odontoblasts to a diet well supplied with minerals and vitamins. It is also possible that proper diet designed to increase the alkaline and calcium contents of saliva, as advocated by Hawkins, plays a part in retarding the carious process in enamel. We do not agree with Bödecker (5) that there is evidence that dental lymph is a factor in preventing or retarding incipient carious processes.

It seems that we have found a sound explanation of the observed phenomenon that natural carious processes very often come to a halt. The lead to our solution of this problem came from a discussion by Beust of Bunting's report at the Louisville meeting of the American Dental Association in 1925. Beust remarked: "If you take a tooth with incipient caries that as yet exhibits no break in surface continuity, and immerse it in lactic acid, you will be surprised to find that the sound enamel will be decalcified and the decayed part remain." This statement, if confirmed, must be given serious consideration in any study of the cause and nature of caries. To determine whether it is correct, thirty entire teeth with incipient caries were immersed in *N* lactic acid for twenty-four hours. Examination of the teeth at this period indicated that Beust's statement was confirmed by twenty-six of the thirty teeth. The areas insoluble in the lactic acid were on the proximal surfaces of the teeth at a place in which caries is usually found. These appeared as flat tables higher than the surface of the more normal enamel, which had been partly dissolved away by the acid (*fig. 5*). The surfaces of the raised areas were sometimes opaque white; at other times, glistening like normal enamel; occasionally they were definitely covered with organic matter visible to the naked eye. In one tooth that had definite cavity formation on one proximal surface, enamel immediately surrounding the cavity was more insoluble even in 5 *N* nitric acid than were other parts of the enamel of the same tooth (*figs. 6* and *7*). In an early experiment twelve teeth with incipient carious areas were exposed to 5 *N* nitric acid. After twenty minutes the teeth showed insoluble areas of enamel having a contour different from that of the "raised areas" resulting from immersion in lactic acid. The nitric acid allowed more sharp-pointed projections to remain on all of the teeth excepting two, on which a demonstration of

an early proximal carious area resulted in an original manner. *Figs. 8* and *9* show front and side views of such an insoluble projection from the proximal area.

The possible connection between these discoveries and a sound explanation of the halt in natural carious processes came to light when we discovered the reason why the incipient carious areas were insoluble in the acid solutions mentioned above. The interesting advance in our knowledge of the cause of their insolubility was made when a tooth showing a projection on each proximal surface after immersion in nitric acid was submitted to the following procedure: One of the insoluble projections was held in a Bunsen flame. Charring took place immediately. The carbonaceous material was completely oxidized by the flame in the presence of oxygen, and the projection examined macroscopically. No change other than the loss of the charred matter was noted in its appearance. However, when the tooth was immersed in the same nitric acid that originally caused the formation of the two projections, the insoluble area that had been freed from organic matter by incineration quickly went into solution, while the other persisted. Considerable further research must be made on the nature of the protective organic matter, but it appears that we have found another demonstration of a well known fact about the human body; namely, that it possesses marvelous means of protecting itself against the inroads of disease. The new demonstration of this fact was particularly evident in the tooth shown in *figs. 6* and *7*, the photographs of which are but poor representations of the marvelous acid-resistant, protecting wall surrounding the cavity.

As to the way in which these insoluble areas of incipient caries of the enamel, or of borders of far-advanced caries, are formed, it seems likely that the acids of fermentation make the enamel more porous by dissolving its more soluble parts, and later these pores become impregnated with organic debris from the mixed saliva. Whether the increased organic material in these carious areas consists of bacteria, parts of bacteria, bacterial products, mucus, or what not, it seems that the ability of the mass to protect the inorganic elements of the enamel from the action of acids explains why the natural carious processes often come to a halt, even when acid-producing bacteria are contiguous to the lesion.

The fact that our evidence thus far largely removes the first two objections to Miller's views, on the importance of acids of fermentation as the active agents in decay of enamel, prompts us to pass quickly over the last three objections. This procedure seems justifiable because Objection 3, concerning the absence of decay on dirty teeth, appears to be removable on evidence like that discussed in relation to Objection 2, and also because Objections 4 and 5 may be only fancied deficiencies in evidence against the part played by local environmental factors.

Objection 3. There has been no satisfactory explanation for the established fact that mouths in a filthy hygienic condition are often free from caries.

In mouths in filthy condition, though free from caries, the teeth are very often dirty owing to tartar deposits of different thicknesses. It is well known that tartar contains a high percentage of tricalcium phosphate, and tends to be similar to enamel in chemical composition. Tartar, of course, is probably more variable in make up than tooth enamel, particularly with reference to its organic content. The discovery of protective organic matter in carious areas indicates that similar depositions result on sound teeth mixed with the inorganic elements of the saliva. And, of course, even if tartar were only pure tricalcium phosphate deposits, it would afford a protection to the enamel against the solvent action of acids of fermentation. Clinical experience seems to support this view.

Objection 4. It is not known why, during pregnancy, caries is more prevalent and makes more progress.

This is one of the most commonly quoted statements to discredit the Miller theory and to support the position of those who maintain that catabolic processes cause decay. We have not found any evidence that dental caries is increased during pregnancy. The aphorism, "For every child a tooth," implying that the mother's teeth are robbed of calcium salts for the benefit of the unborn child, has obtained wide credence. Regarding this popular saying Williams (82) has said: "Whenever I have asked for evidence of this belief based on scientific research, the answer has usually been, 'Oh! that is a matter of common knowledge growing out of everyday experience.' Well, I long ago learned to distrust that sort of knowledge, because I had found out that it is more frequently wrong than right." We agree with Williams

on the unreliability of such evidence. Even if the aphorism were true, the facts do not prove that in pregnancy carious lesions are more numerous or progress more rapidly. In our experience, teeth lost during pregnancy have been those whose enamel and dentin were decayed many months or years before the onset of pregnancy. Williams (82) reports a study by Ziskin at the Minneapolis General Hospital to test this popular belief. Ziskin examined the teeth of 599 women in a pregnant group and 205 in a control group. He came to the conclusion that the aphorism, "A tooth for every child," should be discredited and banished from our minds for all time. Ziskin's findings accord with those of Dogajew (16), who examined 870 teeth from pregnant women, and found no deviation from that of normal caries. Loss of calcium took place from the outer to the inner layers.

The explanation of Naismith (65), and of many other dentists, that rapid decay during pregnancy is due to an absorption of calcium from the enamel to form bones for the fetus, is inadequate for the reasons already given that deficiency in diet is not the active cause of enamel decay in fully erupted teeth; namely, (*a*) there is no circulation in the enamel and (*b*) decay does not occur at all places on enamel, but only at well defined areas. Then, again, the absurdity of such a belief—that the bones of the fetus are in any degree dependent on calcium from the mother's teeth—is obvious from a consideration of the proportion of the calcium in the teeth to that in the bones. Peterson's (67) explanation is a more plausible one; namely, that some change in the oral secretions must account for the rapid decay. In fact, the results of our studies on the effects of acids on enamel, both with and without previous saturation with tricalcium phosphate, considered with results of the analyses of saliva with respect to saturation with tricalcium phosphate (described below), explain how women in pregnancy, with diminished Ca and P contents in the blood and saliva, might have more rapid decay of the enamel.

Objection 5. Caries has not been produced in man under experimental control to substantiate the Miller conception of the etiology of caries.

This is the reason (with the substitution of the word "animals" for "man") that Howe advanced for placing so much importance on deficient diet and defective-mineralization factors. As used by Howe,

the statement carried less weight because the conditions in animals differ from those in man. Consequently, the results with the former are not applicable to the latter. There is danger in proceeding on the false premise that Koch's postulate, as to the need for producing the disease in question with the suspected etiologic agent, must be fulfilled before proof of etiology is complete. We know that this step is unnecessary. Many bacteria associated with different specific diseases have been definitely established and accepted as the causative agents without fulfilling Koch's postulate, because of the lack of satisfactory types of experimental animals.

So many obvious experimental and social obstacles prevent direct bridging of the above mentioned gap, in a well controlled study in man, that other suggestive evidence must be used. We know that the type of microörganism that develops in a medium or substrate, from a mixture of many kinds of organisms, depends on the nature of the medium. A carbohydrate substrate enhances the growth of the aciduric types. The aciduric types produce acidic end-products; the putrefactive or proteolytic types produce basic end-products. In the human mouth there is naturally a mixture of both main types of microorganisms. In different persons the oral and dietary conditions favor, in one case, the deposit of carbohydrate food debris on the teeth; in another case, protein food debris. Millers and bakers who eat (and are exposed to) carbohydrates in large quantities are notoriously subject to dental caries, while Eskimos who subsist wholly on a meat diet are almost free from the ravages of caries. It is not certain that these statements have not grown out of everyday experience, which Williams found so untrustworthy, for we were able to find only a few authoritative references. Hesse (38) writes: "In the Dental Institute of this city (Leipzig) I have had the opportunity of seeing a great number of patients among the industrial and working classes, and have been particularly surprised at the bad condition of the teeth of our bakers. They are affected by caries to such a degree that I have been able, in many cases, since my acquaintance with this phenomenon, to determine the calling of a patient by the condition of his teeth." The information concerning the teeth of Eskimos is more fully reported by Waugh (78, 79, 80) in his studies showing that this race, before it came into contact with our so-called civilized diet, had little or no trouble

with dental caries. Leigh (52) made a careful study of 324 skulls of Eskimos, and corroborated the generally accepted opinion with his finding that only 1 percent had caries. It is possible that some may infer that the lack of decay among Eskimos results from their plentiful intake of vitamin-containing foods, and not alone to the presence of protein-food debris on the teeth. But Stefansson (75), who has lived with the Eskimos for a longer period than any other white man, claims that the diet of Eskimos who have not come into contact with civilization is very deficient in vitamins, as their diet is practically always the muscular part of fish. It is not intended to suggest, in this discussion, that a carnivorous diet is essential to the prevention of tooth decay. Only recently McCollum (56) has called attention again to the fact that people on a vegetarian diet, or on a vegetarian diet supplemented by milk, have in general distinctly better teeth than have Americans.

From these considerations of the strong points and shortcomings in the views of the two foremost schools of thought, we came to the conclusion that a study designed to fill in the gaps and extend the boundary of knowledge of the local environmental effects might be successful in demonstrating the true nature of the *active* carious process in enamel. It seems evident that any attempt to broaden our knowledge of the local environmental factors in the etiology of dental caries should include investigations of (*a*) the effects of acids on enamel; (*b*) a bacteriological survey of carious and non-carious teeth; and (*c*) the relation of saliva to the carious process. There are many important subdivisions of these three main subjects. For the sake of clarity, these will be noted casually in the presentation of the results.

IV. STUDIES OF THE EFFECTS OF ACIDS ON ENAMEL

A. Discussion of previous work in this field

One of the greatest obstacles in the way of testing Miller's conception of the etiology of dental caries is the limited amount of reliable fundamental information on the effects of acids on enamel. In order to demonstrate the lack of agreement in results even in studies that have been made within the last decade, a few of the recently published statements will be reported. Rodriguez (71) noted that teeth suspended in dilute lactic acid (pH 3.9) showed no observable change in

ten weeks. But he did find dullness, localized softened areas, or translucency, at the end of twelve weeks in teeth that had been subjected in a medium to the action of acids produced by bacteria that gave a final pH of 3.9 to 3.6 in the medium. McIntosh (57), in a study of the effects of acid nutrient-broths (pH 5.0 to pH 1.0) for thirty-four weeks, reported that enamel was attacked at pH 1.0, pH 2.0, and pH 3.0, but not at pH 4.0, 4.5, or 5.0. Hartzell and Larson (34) reported that a lactic acid solution of pH 2.5 did not seem to attack teeth, but they noticed white decay at pH 7.5 in the presence of Aspergillus niger in broth. Bunting and Palmerlee (8) noted whitening of the teeth in solutions of lactic acid at pH 2.5, 3.8, and 4.0 in eleven days, and at pH 4.8 and 5.0 in four and one-half weeks. The same authors found that aciduric organisms in glucose media produced whitening of enamel in from six to eight days. The final pH of the media was 3.4–3.6. McClelland (54), by the use of the gravimetric method, found that enamel was not decalcified in buffered solutions between pH 6.5 and pH 11.0 during sixty-two days. In solutions more acid than pH 6.5, he observed that enamel was decalcified at a speed proportional to the degree of H-ion concentration. At pH 11.5 and 12.0, slight loss of weight was noted. Much of the value in McClelland's report is lost because he did not give the composition of his buffer solutions. One of the latest reports is that of Thurlow and Bunzell (76). While they picked flaws in the methods of McClelland (54) and Pickerill (68), and others who give results in terms of loss of weight, the value of their own results is impaired by the deficiency of the method they chose to ascertain the effects of acid on enamel. They determined the percentage loss of enamel, calculated from the relative weight of enamel to the whole tooth, for which they had to measure the surface, thickness, and density of the enamel. Their own appraisal of this method was stated as follows: "This procedure, as will be seen, can aim at giving only a rough idea of the percentage weight of the enamel, as so many experimental errors are involved."

The work of Friesell and Vogt (20), on the effects of acids on enamel, though narrow in scope, seems to furnish the most reliable data available today.[9] In their tests of the action of dilute acids on enamel, the

[9] Since this paper was written, Benedict and Kanthak have published an important contribution on this subject: *Journal of Dental Research*, 1932, xii, p. 277.

solutions were made to flow slowly over the tooth. The rate of flow was so slow that, while the solution was constantly removed from the vicinity of the tooth, there was no danger of wearing the tooth by any *mechanical* action of the solution itself. Distilled water, freed from carbon dioxide, produced opaque spots on the enamel that could be detected macroscopically by the end of eight weeks. A definite etching of the enamel was observed at the end of six weeks when lactic acid was used in the following dilutions: 1 part of acid in 110,000 to 140,000 parts of water (CO_2 free); 1 part of acid in 500,000 to 700,000 parts of water; and 1 part of acid in 1,100,000 to 1,400,000 parts of water. The pH values of these solutions, determined by the authors, were respectively 4.0–4.1, 4.65–4.8, and 5.0–5.1. Water fully carbonated at atmospheric pressure caused several opaque white spots in two days, while the entire surface of enamel was opaque at the end of the third day. Carbonated water saturated with tricalcium phosphate did not etch enamel in eight weeks. After twelve weeks, no appreciable etching could be detected on the surface of enamel immersed in solutions at pH 7.2 or pH 7.4, even though these solutions contained no tricalcium phosphate.

Many pitfalls must be avoided in the choice of experimental procedure for a study of the action of acids on enamel. The most common fault in previous experimentation has been the deficiency whereby a constant H-ion concentration has not been maintained. There are several ways of removing this defect as follows: (*a*) a method in which a continuous stream of acid is allowed to trickle over the enamel; (*b*) the method used by McClelland, whereby a system of buffer solutions is used to maintain a constant pH; and (*c*) a method of exposing an enamel area so small that the products of the reaction between the exposed enamel and the buffer solutions will be present in quantities too slight to affect the reaction of the solutions to a practical degree. While the last two methods are not quite as efficient as the first, they are much less cumbersome and better adapted to a broad study of the action of acids on enamel. Another important defect in the experiments of many investigators of this problem was the use of the gravimetric method to determine the effects of acids on enamel. This method appears to be fundamentally limited in value because it postulates that injury to the enamel is accompanied by a proportionate loss

of weight. A macroscopic or microscopic examination of either natural or artificial carious lesions shows the fallacy of such an assumption. Enamel may be seriously damaged by acid without loss of weight within the limits of experimental error, as evidenced by loss of polish, increased opacity, and chemical change, *e.g.*, from a tri- to a di-calcium phosphate. The most delicate method of detecting the effect of acids on enamel is the microscopic method, particularly if sections are stained with silver nitrate to indicate increased porosity of the enamel. The grinding and mounting of as many tooth sections as are needed in a broad study of this nature is a vast and tedious program, but the importance of the information to be gained seemed to justify the undertaking in the experiments described below.

B. The effect of simple lactate- and citrate-buffer solutions on tooth enamel

The teeth used in this experiment were preserved, in great masses, in formalin (4 percent) for various lengths of time. The significance of the term "great masses" should be noted. If the teeth had not been preserved in a solution previously saturated with the elements in the teeth, it is likely that our experimental study of the effects of acids on enamel would be less reliable. In other words, pure water or sodium chloride solution in adequate volume would dissolve the enamel sufficiently to be detected with the aid of a microscope. Two hundred and fifty-two (252) incisors with apparently perfect labial surfaces were selected from the large number of teeth preserved in the formalin solution. It was decided to cover all the surface of each tooth except one small area. After preliminary experimentation, there was chosen for this purpose a special grade of asphaltum, which was free from acid, had a high melting point, and adhered tenaciously to the tooth surfaces. After the teeth were carefully covered with the asphaltum, an area, slightly smaller than a cross-section of the head of a common pin (1.5 sq. mm.), was exposed at a place on the labial surface chosen for the excellent condition of its enamel. Sketches were made of all the teeth showing the exact location of the exposed areas.

Buffer solutions were prepared in the following manner: Sufficient 0.2 *N* sodium hydroxide was added to 0.2 *N* lactic acid to produce the following H-ion concentrations (pH): 4.0, 4.6, 5.0, 5.2, 5.4, 5.6,

and 5.8; and to 0.2 *N* citric acid, to produce (pH) 5.0, 6.0, 6.2, 6.4, 6.6, 6.8, 7.0, 7.2, 7.4, 7.6, and 8.0. It was desirable to have the lactate buffer cover the whole range, but this solution loses most of its buffer action above pH 5.0. All pH determinations were made by means of the colorimetric method. Each tooth, covered in the manner described above, was immersed in 40 cc. of buffer solution, and one tooth was removed from each of the different solutions at the end of the following periods: 1, 2, 3, 4, 5, 7, 9, 11, 14, 21, 30, 40, 50, and 60 days.

The teeth were freed from asphaltum with the aid of toluol and inspected macroscopically for indications of localized etching. These gross observations gave the information that tooth enamel was etched in each buffered solution from pH 4.0 to pH 8.0, especially during the longer periods of immersion. As a more detailed and accurate picture of the effects is obtainable from the stained sections through the etched areas, further discussion of the macroscopic observations will be omitted from this report.

The final pH of the buffer solutions in which the teeth were immersed was always determined. The H-ion concentration of each buffer solution was found to have remained practically constant, except for a slight shift of 0.2 pH to the alkaline side in the solutions that had acted on enamel for the longer periods of time. There was also observed a greater exception to this constancy of H-ion concentration in the lactate buffers of pH 5.6 and 5.8 that acted on teeth for periods of two weeks to sixty days. The final pH in these buffer solutions ranged between 6.4 and 7.4. The greater shift is easily explainable on the basis of the negligible buffer effect residing in the pH 5.6 and 5.8 lactate-buffer solutions for additional amounts of alkali.

The staining procedure in the *first* experiment on the effect of acids on enamel was as follows: The entire tooth from which the asphaltum had been removed was immersed, for seventy-two hours in the dark room, in a freshly prepared 5 percent silver nitrate solution (neutral to litmus). The tooth was then washed in running water, still in the dark, until washings showed no precipitation on the addition of sodium chloride solution (10 percent). Most of the water was poured off, allowing only enough to remain to prevent the tooth from getting dry, and the bottle containing the tooth was set in the light. This staining

method was unsatisfactory, because so much reduced silver developed, even on the control surface of the enamel, that later grinding carried a considerable part of it to greater depths, making the differentiation of control and etched areas quite difficult except where the solvent action of the acid was pronounced. In the *second* experiment on the effects of acid on enamel, we made certain modifications in the staining procedure that gave much more satisfactory results. The changes in technic dealt with the method of washing the excess of silver from the tooth. We first used a sodium chloride solution, then a sodium thiosulfate solution, and finally running water. When a tooth subjected to such a procedure was exposed to light, it was seen that the staining was confined almost entirely to that area which had been acted upon by the buffer solution.

After staining each entire tooth with silver nitrate, they were ground through the etched areas into thin longitudinal sections perpendicular to the labial enamel surfaces. The grinding of sections was begun with unsatisfactory types of stone, only 167 of the 252 ground teeth withstanding this treatment. Subsequently Dr. J. Leon Williams kindly furnished two grinding stones and many valuable suggestions that made it possible for later sections to show to better advantage, both as to appearance and number of teeth successfully ground. The sections were mounted on glass slides and examined microscopically. Photomicrographs of the mounted sections were arranged on a chart vertically according to periods of immersion, and horizontally according to H-ion concentration of the buffer solution that etched them. *Table 1* gives this record of the effects of the simple lactate-and citrate-buffer solutions on tooth enamel. These observations were made on the sections magnified 40 times. It would be impracticable to include all the pictures in this report. It seems desirable, however, to present (*a*) a group (Series 1) showing the effects of a simple lactate-buffer solution (pH 4.0) for various times; (*b*) a group (Series 3), the effects of a simple lactate-buffer solution (pH 5.0); and (*c*) a group (Series 5), the effects of a buffer solution of pH 6.8 resembling that of saliva, i.e., around the neutral point. The reasons for recording these three series will be obvious after an examination of the results of our further experiments on the action of acid on enamel.

Three important advances in our knowledge of the effect of acids on enamel resulted from this part of the investigation in which simple lactate- and citrate-buffer solutions were used; namely, (*a*) certain ground sections of artificial caries were found which so closely simulated natural caries that any dental histologist would experience difficulty in deciding whether they were sections of artificial or natural caries; (*b*) not only was Friesell and Vogt's contention—that solution of pH 7.0

TABLE 1

*Microscopic effects of simple buffer solutions on enamel**

DAYS	CITRATE BUFFERS (pH)											LACTATE BUFFERS (pH)						
	5.0	6.0	6.2	6.4	6.6	6.8	7.0	7.2	7.4	7.6	8.0	4.0	4.6	5.0	5.2	5.4	5.6	5.8
1	3	2	2					2			1	4		3		2	3	
2		4	2			1		2	2	3		4		3		3	3	
3						2				3		3		3	5		3	
4	3		2	2	1	2	2					5	5					
5	4		3	2	2		3		2			6	5	4	4			1
7					2		3	2	2	2	3		5	4				
9	5	2	3	3		3	2		2	2		6	5	4	3			4
11	4	3	3		4	2		4	2		1		6	5	4	2	4	3
14	5		3			3	4	3	2		4	6	6		4	3	3	3
21	5	3	4	3	3	4		3	4		2	7	5			4	4	4
30	7	3	4	4	2	5	5	4	3			6		4	6			3
40	7	4	4		3	5		3	4		3	6		6	4	3	4	4
50	6	4	5	3	4	5	5	4	4	3	4			6	6	4	4	3
60	7	5	5	4	4	4	4	4	4	4	4		7	5	5	3		6

* Explanation of the numerals: 1 = Enamel not affected by the solution. 2 = Very superficial etching of enamel. 3 = Marked etching, without loss of surface continuity. 4 = Marked etching, with slight loss of surface continuity. 5 = Marked etching, with greater loss of surface continuity. 6 = More pronounced 5-effect. 7 = Loss of enamel to dento-enamel junction.

could etch enamel—corroborated, but conclusive evidence was also obtained that each of our buffer solutions from pH 4.0 to pH 8.0 etched enamel, especially during the longer periods of immersion; and (*c*) although, as a general rule, the greater the H-ion concentration of the buffer solution and the longer the period of immersion of the enamel in the solution, the more pronounced the solvent effect, there were sufficient exceptions to this rule to indicate that different enamels vary in their susceptibility to the action of acids.

C. Relation of the Ca, P, and H-ion concentration of the saliva, and of the solution in direct contact with a carious process, to the progress of caries

The experiment described above demonstrated that eighteen simple $\frac{\text{lactic acid}}{\text{sodium lactate}}$ and $\frac{\text{citric acid}}{\text{sodium citrate}}$ buffer-solutions from pH 4.0 to pH 8.0 injured enamel definitely in seven days, and in a most pronounced manner during thirty to sixty days. It is obvious that this information does not indicate how the different strengths of organic acids affect teeth in the mouth. If it did, human teeth would not exhibit even the degree of durability that they do. It also is obvious, on the principle of the common-ion effect, that the following evidence, that saliva is saturated with tricalcium phosphate, is an important link in the explanation of the protection of teeth in the mouth from injury by solutions neutral in reaction or even slightly acid or alkaline. A comprehensive understanding of the action of tricalcium phosphate in solution is necessary before calculations or deductions concerning the concentration of this salt in saliva can be made. The best source of such information known to the authors is the "Studies in Calcification" by Holt, LaMer and Chown (40). The following is a brief résumé of the principles underlying their experimentation and calculations.

Tricalcium phosphate cannot precipitate from solution unless the ion product, $(Ca^{++})^3 \times (PO_4^{=})^2$, exceeds the equilibrium value, K s.p. If the quantities of calcium and phosphorus are determined, a knowledge of the pH makes it possible to calculate the concentration of $(PO_4)^{=}$ ion and then the solubility product constant of tertiary calcium phosphate. K s.p. (solubility product constant) $= (Ca^{++})^3 \times (PO_4^{=})^2$ in systems that are in equilibrium with solid tri-calcium phosphate. The true or thermodynamic product for tricalcium phosphate, which is a constant under all conditions, is a product of the activities or active masses of Ca^{++} and $PO_4^{=}$ ions, and may be written:

$$(a\ Ca^{++})^3 \times (a\ PO_4^{=})^2 = a\ K\ s.p.$$

No methods, however, are available to measure individual activities, so we are confined to the stoichiometric solubility product, K s.p. Owing to the unwieldiness of this quantity in calculation, it is usually

replaced by the p K s.p., which is the negative logarithm of K s.p. Therefore the lower the value of the p K s.p., the greater is the ion product. Holt, LaMer and Chown's experiments showed that the K s.p. is greatly affected by salts and proteins, but is constant in a solution of constant concentrations of these other substances. They determined the K s.p. of tricalcium phosphate in serum by shaking 190 cc. of serum with 10 cc. of a solution of various amounts of calcium and phosphate, and precipitated the tricalcium phosphate until the system came to equilibrium before determining the calcium and phosphorus in solution. Their results indicated that the solubility product in blood serum is about 1×10^{-26} (p K s.p. = 26.0), as compared with a p K s.p. of 32.5 in pure water.

R. B. Smith (74) calculated an approximate p K s.p. for tricalcium phosphate in saliva. In his report he wrote: "A solution which is isotonic with the blood contains 0.85 to 0.90 percent of sodium chloride, and saliva contains about 0.2 percent of total solids. Since saliva contains only about one fourth the total solids of blood serum, the solubility of tricalcium phosphate should be less than in blood serum. Holt, LaMer and Chown have shown, from theoretical considerations,

$$\Delta \text{ p K s.p.} = 15 \sqrt{M}$$

where M is the activity coefficient of the soluble salt, and $\Delta\ p\ K\ s.p.$ is the change in the p solubility product. They have tested this formula, using the inorganic salts of blood serum and five other salts, and have found that the experimental results are in good accord with the calculated results. Using this equation, it was calculated that p K s.p. of tricalcium phosphate in saliva should be approximately 29.5." This takes into account only the inorganic salts dissolved in saliva. The effect of the protein is not considered. Holt, LaMer and Chown made an attempt in this direction by determining the p K s.p. of tertiary calcium phosphate in a solution containing only the inorganic salts of serum. They found it to be 27.2, while it was 26.0 in actual serum that contained not only these inorganic salts, but also from 7 to 8 percent of protein. While the valence of protein ions is so large that one might expect a much more striking difference, the molecular weights of the serum proteins are so high that the actual molar concentration of protein must be very small. The concentration

of mucin, the protein in saliva, as given in Matthews' *Physiological Chemistry* (53) from Frerich's analysis, is 0.213 per cent. It is very unlikely that this small amount of protein could have any effect that need be considered when an amount 35 times greater in serum has an effect that is very slight. The authors, then, in the light of available information, accept 29.5, the quantity calculated by R. B. Smith, as the probable p K s.p. of tricalcium phosphate in saliva.

Turning to the results of Vogt's (77) salivary analysis, as recalculated by Smith: there is not one case among 225 that had complete figures for calcium, phosphorus and H-ion concentration, in which the p K s.p. was higher than 28.81; the ion product, therefore, is always greater than that required for saturation. The average values for the p-product of four different groups (arranged according to condition of teeth) are 25.91, 26.02, 26.13, and 26.26, starting with the group that had little or no caries and ending with the group that had considerable caries. All these results indicate not only a state of saturation but one of supersaturation. If this is true, if the saliva is supersaturated with tricalcium phosphate, why is there not a precipitation of the salt in excess of that required for saturation? All through their papers these workers show that the precipitation of tertiary calcium phosphate is a slow process. Holt (40), studying the equilibria concerned with the calcification of bone in *persons having rickets*, found that the serum was supersaturated with tricalcium phosphate. The degree of supersaturation was not as great as that in cases where the disease was absent or healing. From this it would seem that the degree of supersaturation is a factor of great importance in the deposition of the salt. Another investigator in this field, Csapo (15), in reviewing the influence of proteins on the solubility of calcium phosphate, said that proteins inhibit a deposition of calcium salts which other physical factors tend to produce. At any rate, whatever the cause, it is the general observation of all these investigators that tertiary calcium phosphate shows a tendency to remain supersaturated in solution for long periods.

The above statements would help to explain conditions if saliva, supersaturated with tricalcium phosphate, remained in contact with the solid salt in the form of tooth enamel, without any precipitation. There is, however, considerable evidence indicating true deposits of tricalcium phosphate on the teeth. The so-called calculus or tartar

formation on the teeth is composed almost entirely of calcium phosphate, with some calcium carbonate, mucin, epithelial cells, and organic débris from the food. It is highly improbable that this calcium phosphate could come from any other source than saliva. Prinz (69), while studying the formation of tartar on the roots of teeth, was able to obtain a tartar formation from natural saliva outside the mouth. He placed a series of teeth in embedding material and loosened them a little. They were kept in an incubator at 40°C., and the artificial pockets around them filled daily with fresh saliva. Within eight to ten weeks these teeth were removed from the embedding material and presented typical tartar incrustations upon their roots which, in every respect, were identical with those formed on teeth in the human mouth. He then prepared an artificial saliva agreeing with the analysis of Hammerbacher (31), using as a colloidal menstruum for the preparation a solution of 2.5 grams of powdered albumin in 1000 cc. of water. A stream of carbon dioxide was passed through the solution to dissolve the calcium and magnesium phosphates. This solution was kept on ice and used in an experiment like the one described above, except that the solution in the pockets about the teeth was changed several times a day. The tartar incrustations that formed upon the roots of the teeth in this experiment were identical with those obtained from natural saliva out of the mouth and with those found upon teeth in the mouth. Another investigator in this same field, McClendon (55), in his article on "The Equilibrium Between the Enamel of the Teeth and the Saliva," describes work with apatite, $CaF,Ca_4(PO_4)_3$, a mineral very similar in composition to tooth enamel. At pH between 5.5 and 6.5 the apatite was practically at equilibrium with an artificial saliva containing the same quantity of calcium phosphate as that in normal saliva. From this, and the fact that impure masses of calcium phosphate deposit on the teeth, he concludes that, if the surface of the enamel is sufficiently cleaned theoretically the enamel should receive deposition of calcium salts with increase of pH and dissolve with a decrease of pH.

The two main points, then, taken as evidence that tricalcium phosphate is present in the saliva at a concentration exceeding that required for saturation, are the following:

(a) Analysis shows that the ion product exceeds the calculated equilibrium value or solubility product constant (K s.p.).

(b) There is a deposition of solid tricalcium phosphate in the so-called tartar precipitated from saliva on teeth.

While this evidence—that in the saliva $(Ca^{++})^3 \times (PO_4^{=})^2$ generally exceeds the equilibrium value, K s.p.—is probably helpful in showing why those diseases in which the entire enamel-crowns of teeth dissolve away are not more common, the discussion thus far does not aid particularly in a consideration of the mechanism of the carious process. In this disease, characterized especially by its localized nature, there are particular areas in the mouth where the $(Ca^{++})^3 \times (PO_4^{=})^2$ conditions, in the solution in direct contact with the enamel of pits, fissures, and proximal and gingival areas, differ from the $(Ca^{++})^3 \times (PO_4^{=})^2$ conditions of the general saliva. This difference is caused by plaques, which prevent free access of saliva to the organic acids produced by the fermentation of carbohydrate débris. It is probably impossible to determine accurately the specific conditions in the solution adjacent to any localized area of enamel with respect to calcium and phosphorus concentrations. It would be "fictitious accuracy" if we could, because our theoretical discussion is based on the assumption that enamel is pure tricalcium phosphate and, of course, this is not true. There are so many variations in enamel and saliva, however, that, since enamel is composed of inorganic salts primarily and the keratin content—determined by Rosebury (73)—averages only 0.42 percent, we may assume that the solution of enamel follows the same laws as that of any other very insoluble compound. As analyses of the enamel show that it consists of about 90 percent of tricalcium phosphate, we may assume for this discussion that it is pure tricalcium phosphate, feeling that what holds true for this salt will apply with limitations to tooth enamel. A recent contribution by Forbes (19) makes it unnecessary for us to describe dental caries in more detail from a physico-chemical standpoint.

D. The effect on tooth enamel of lactate- and citrate-buffer solutions previously saturated with tricalcium phosphate

Our main purpose here is to report an experiment on the action of acids on enamel under conditions that, we believe, approximate more closely those of natural caries than any that have heretofore come to our attention. In the solution underneath plaques we may assume, as described by Forbes, that there is a saturated condition with

respect to tricalcium phosphate, but that, as lactic acid is produced by fermentation, there is a decrease in $PO_4^{\equiv}$ concentration owing to the formation of $HPO_4^{=}$ and $H_2PO_4^{-}$ ions as the H-ion concentration of that particular locality increases. With the diminution of $PO_4^{\equiv}$ concentration, a condition exists immediately adjacent to the enamel where temporarily $(Ca^{++})^3 \times (PO_4^{\equiv})^2$ is less than K s.p. The effort to come to equilibrium is then responsible for the solution of the enamel. This continues as long as more H ion is produced in the locality, so that in the solution immediately adjacent to an active carious lesion, the calcium, phosphorus and H-ion concentrations represent values where $(Ca^{++})^3 \times (PO_4^{\equiv})^2$ is less than K s.p.

In order to make this second part of the study of the effects of acids on enamel comparable to our previous study, in which simple $\frac{\text{lactic acid}}{\text{sodium lactate}}$ and $\frac{\text{citric acid}}{\text{sodium citrate}}$ buffer-solutions were used, and also more nearly comparable to natural conditions, the buffer solutions for this latter investigation were prepared in the following manner: Simple buffer-solutions were made as in the previous study, by adding the required amount of $N/5$ sodium hydroxide to definite quantities of $N/5$ lactic acid and $N/5$ citric acid, to produce various H-ion concentrations ranging from pH 4.0 to 8.0. To each of the buffer solutions was added an excess of finely divided powdered tricalcium phosphate. These solutions were shaken continuously for three days; by that time the pH values had shifted to the alkaline side generally about 0.4 of a pH unit. Although it is likely that a 3-day period is not sufficient for thorough saturation and attainment of equilibrium with respect to tricalcium phosphate, it was believed that the conditions sufficed for our purpose. After the 3-day contact with the tricalcium phosphate, the supernatant solutions were filtered. The required amount of $N/5$ acid, either lactic or citric, was added to each buffer solution to bring it back to the original pH. These buffer solutions were then allowed to stand with occasional shaking for fifteen days until they were at equilibrium, as determined by H-ion determinations. The pH values of these buffer solutions previously saturated with tricalcium phosphate are recorded in *tables 2* and *4*.

As a preliminary to the more-time-consuming experiment designed to show the microscopical effects on localized areas of enamel of

different buffer solutions previously saturated with tricalcium phosphate, it seemed advisable to get a definite clue by the more simple

TABLE 2

*Effects of simple buffer solutions on enamel**

ORIGINAL pH	BUFFER	TEETH NO.	PERIOD OF IMMERSION					FINAL pH
			24 hrs.	48 hrs.	72 hrs.	96 hrs.	17 days	
4.0	Lactate	1	++++	++++	++++	++++	++++	4.3
4.0	"	2	++++	++++	++++	++++	++++	4.2
4.4	"	1	++++	++++	++++	++++	++++	4.6
4.4	"	2	++++	++++	++++	++++	++++	4.7
4.8	"	1	+++	+++	+++	+++	++++	4.9
4.8	"	2	+++	+++	+++	+++	++++	5.0
5.2	"	1	+++	+++	+++	+++	++++	5.2
5.2	"	2	+++	+++	+++	+++	++++	5.4
4.0	Citrate	1	++++	++++	++++	++++	++++	4.3
4.0	"	2	++++	++++	++++	++++	++++	4.2
4.8	"	1	+++	+++	+++	+++	+++	5.0
4.8	"	2	+++	+++	+++	+++	++++	5.0
5.2	"	1	+++	+++	+++	+++	++++	5.4
5.2	"	2	+++	+++	+++	+++	++++	5.4
5.6	"	1	+++	+++	+++	+++	++++	5.8
5.6	"	2	+++	+++	+++	+++	++++	5.8
6.0	"	1	+++	+++	+++	+++	++++	6.2
6.0	"	2	+++	+++	+++	+++	+++	6.2
6.4	"	1	+++	+++	+++	+++	++++	6.6
6.4	"	2	+++	+++	+++	+++	+++	
6.8	"	1	+++	+++	+++	+++	+++	7.0
6.8	"	2	+	+	+++	+++	+++	7.0
7.2	"	1	+	+	+++	+++	+++	7.4
7.2	"	2	++	+++	+++	+++	+++	7.4
7.6	"	1	+−	+−	+	+	+	8.4
7.6	"	2	+	+	+	+	+	8.4
8.0	"	1	−	−	−	−	−	8.4
8.0	"	2	−	−	−	+	+	8.4

* Explanation of the symbols: ++++ = Marked solvent action, causing the formation of a chalky powder that could be easily wiped off the tooth. +++ = Marked whitening effect and loss of polish. ++ = Marked whitening effect; luster still persists. + = Slight etching and slight loss of luster. +− = Very slight indication of cleansing effect only; luster still persists. − = No visible effect.

macroscopical method—to learn whether the evidence to be obtained from the more tedious procedure would be valuable in helping to solve our problem. Fifty-six incisors were selected for the relatively good

condition of their enamel. Each was attached at the root to a short piece of narrow glass-tubing, which was secured in a perforation in a cork stopper so that it could be suspended in a bottle. In this test we used both simple buffers, and buffers previously saturated with tri-

TABLE 3

*Effects on enamel of buffer solutions previously saturated with tricalcium phosphate**

ORIGINAL pH	BUFFER	TEETH NO.	PERIOD OF IMMERSION					FINAL pH
			24 hrs.	48 hrs.	72 hrs.	96 hrs.	17 days	
4.2	Lactate	1	−	++	++	++++	++++	4.2
4.2	"	2	−	+−	++	++++	++++	4.2
4.6	"	1	+−	+−	++	++	++	4.6
4.6	"	2	−	−	−	−	+++	4.6
5.0	"	1	−	−	−	−	−	5.0
5.0	"	2	−	−	−	−	−	5.0
5.4	"	1	−	−	−	−	−	5.4
5.4	"	2	−	−	−	−	−	5.2
4.2	Citrate	1	++	++	++++	++++	++++	4.2
4.2	"	2	++	+++	++	++++	++++	4.2
4.9	"	1	+−	+−	++	++	++	5.0
4.9	"	2	+−	+−	+−	+−	+++	5.0
5.4	"	1	−	−	−	−	−	5.5
5.4	"	2	−	−	−	−	−	5.6
5.8	"	1	−	−	−	−	−	5.8
5.8	"	2	−	−	−	−	−	5.8
6.1	"	1	−	−	−	−	−	6.2
6.1	"	2	−	−	−	−	−	6.2
6.6	"	1	−	−	−	−	−	6.7
6.6	"	2	−	−	−	−	−	6.8
7.0	"	1	−	−	−	−	−	7.0
7.0	"	2	−	−	−	−	−	7.1
7.4	"	1	−	−	−	−	−	7.2
7.4	"	2	−	−	−	−	−	7.6
7.8	"	1	−	−	−	−	−	8.0
7.8	"	2	−	−	−	−	−	8.0
8.2	"	1	−	−	−	−	−	8.4
8.2	"	2	−	−	−	−	−	8.4

* The symbols are explained in *table 2*.

calcium phosphate. Two teeth were run in each solution. Fifty-six bottles (in two sets of twenty-eight) each received 90 cc. of the different solutions, in which the teeth were immersed in the solutions to a depth of $\frac{1}{8}$ inch on the crown; allowed to remain for seventeen days;

and examined daily during the first four days. At the end of this time the teeth were removed from the solutions, examined, attached to cards, and labeled. It was found that the final H-ion concentrations of the solutions had remained relatively constant. The detailed observations are recorded in *tables 2* and *3*.

TABLE 4

*Microscopic effects on enamel of buffer solutions previously saturated with tricalcium phosphate**

DAYS	TOOTH	CITRATE BUFFERS (pH)										LACTATE BUFFERS (pH)			
		4.3	4.4	4.8	5.6	6.0	6.4	6.8	7.2	7.8	8.0	4.0	4.3	4.6	5.0
1	Upper	3		2	2	2	1	2			1	2	2	2	1
	Lower	2	3	2	1	2		2	2	2		3	2	2	1
7	Upper	3	4	3	1	1	2	2		2		5	4	4	1
	Lower	3	3	2	2	1	1	2	2	2	1	5	3	4	
14	Upper	3	3	3		1	2		2	2	1	5	3	3	2
	Lower	4	4	3	2	1	2	1	2		1	5	3	3	2
21	Upper	3			3	2	2	2		2	1	5	3	3	
	Lower	4	3	3	2	2	2	2	1	2		5	5	3	2
30	Upper	2	3	3	2		2	2	2	2	1	3	5	3	1
	Lower	2		3	2	2	2	2	1	2	1	5	5	3	2
40	Upper	3	4	3	3	1	2	3	2	1		5	5	5	
	Lower	4	3	3	3	2	1		1	1	1	5	5	5	2
60	Upper	3	4	3	3	1	1	2		2	2	5		4	2
	Lower	3	5	3	2	2	2	2	1	2		7	3	4	2

* Explanation of the numerals: 1 = Enamel not affected by the solution. 2 = Very superficial etching of enamel. 3 = Marked etching, without loss of surface continuity. 4 = Marked etching, with slight loss of surface continuity. 5 = Marked etching, with much loss of surface continuity. 6 = Marked etching, with slightly greater loss of surface continuity. 7 = Loss of enamel down to dento-enamel junction.

The comparative data in these two tables show that the protective action of Ca^{++} and $PO_4^{=}$ ions is very effective at pH 5.0 and above. This finding is so important that it was necessary, before relying upon it, to turn to the more refined technic of microscopical examination of ground sections through the etched areas. The importance of the

discovery lies in the fact that organisms incapable of producing or tolerating a pH less than 5.0 in the mouth could be excluded as potential causes of caries of the enamel.

For the microscopic examination of exposed areas, the teeth were covered in the manner described on page 789. It was decided to expose an upper and a lower incisor to each solution for each period of time, to test the popular belief that upper teeth are more susceptible to caries than are the lowers. These teeth were carefully chosen for the good condition of their enamel. A separate bottle was used for each solution for each pair of teeth for each period of immersion, as shown in *table 4*. The different periods of immersion were 1, 7, 14, 21, 30, 40, and 60 days. After the teeth had been stained, sections were prepared, mounted, examined, photographed, and the pictures arranged on a chart as described previously. The picture of the upper tooth was placed directly above that of the corresponding lower one.

It is practical to present here only the photomicrographs (*figs. 10, 11,* and *12*) corresponding to the series showing the effects of the simple buffer-solutions. Series 2, 4, and 6 are comparable with Series 1, 3, and 5, respectively. The protective effects of the tricalcium phosphate in the acid solutions saturated with it, compared with the effects of the acid solutions that did not contain tricalcium phosphate, as shown for the pH 5.0 and 6.8 groups (*figs. 11* and *12*), are similar to the protection afforded by the calcium phosphate in all H-ion concentrations ranging from pH 5.0 to pH 8.0. On the other hand, the pH 4.0 group of photographs (*fig. 10*) shows that there is no protection at this acidity, a finding that coincides with the results observed at pH 4.3 and pH 4.6.

The results of the microscopic examinations of the sections exposed to buffers previously saturated with tricalcium phosphate are shown in *table 4*. They should be compared with the results in *table 1*, showing the microscopic effects of the simple buffers. These tables reveal that all the simple buffers etch enamel to a considerable extent, especially in the longer periods. Of the buffer solutions previously saturated with tricalcium phosphate, however, none of those at pH 5.0 or above etched enamel, except to the most superficial degree, even during 60-day immersions.

We exposed one upper and one lower incisor to each buffer for each

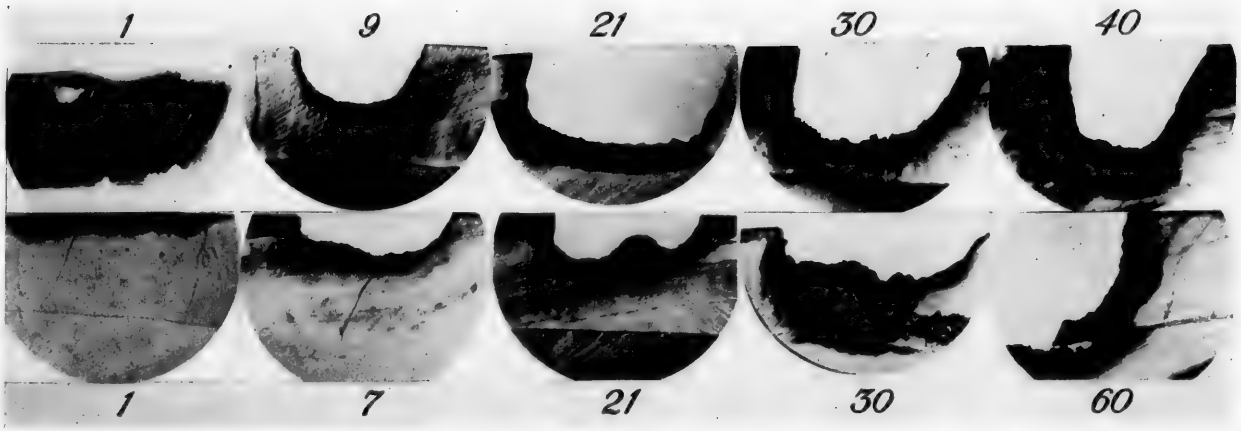


FIG. 10. Effects of lactate-buffer solutions (pH 4.0) on enamel. ×20
Upper row: Series 1—not previously saturated with tricalcium phosphate.
Lower row: Series 2—previously saturated with tricalcium phosphate.
Marginal numerals indicate respective number of days of immersion.

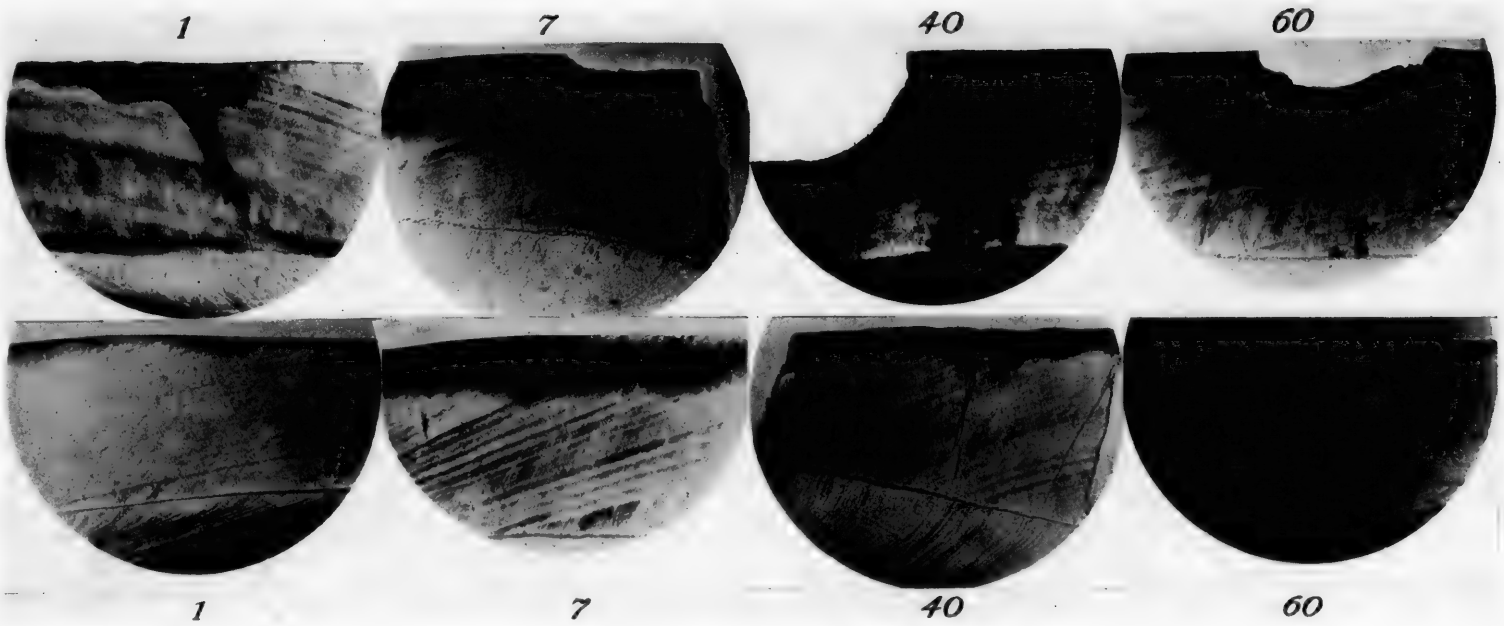


FIG. 11. Effects of lactate-buffer solutions (pH 5.0) on enamel. ×30
Upper row: Series 3—not previously saturated with tricalcium phosphate.
Lower row: Series 4—previously saturated with tricalcium phosphate.
Marginal numerals indicate respective number of days of immersion.

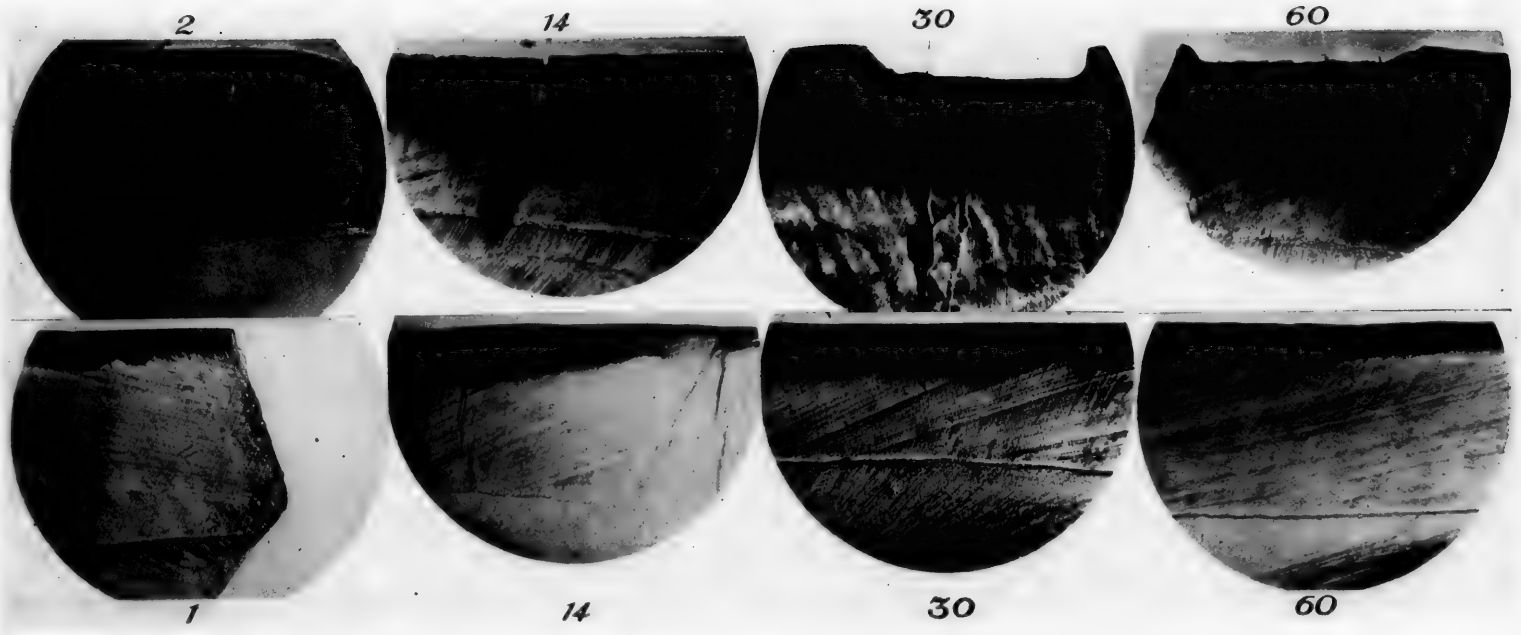


FIG. 12. Effects of citrate-buffer solutions (pH 6.8) on enamel. ×30
Upper row: Series 5—not previously saturated with tricalcium phosphate.
Lower row: Series 6—previously saturated with tricalcium phosphate.
Marginal numerals indicate respective number of days of immersion.

period; therefore it has been possible to compare the relative susceptibility of 69 pairs of uppers and lowers. Of these teeth, 14 showed greater etching on the upper, 19 on the lower; and in the remaining 36 pairs, the upper and lower were etched equally. Since, as noted in a former experiment, teeth from the same mouth differ in their solubility in acids, a conclusive statement as to the relative susceptibility of uppers and lowers to the action of acids will have to be withheld pending research on pairs of teeth from the same mouth.

E. Summary of effects of acids on enamel

(a) Certain ground sections of artificial caries so closely simulated those of natural caries that dental histologists experienced difficulty in deciding whether the slides represented artificial or natural caries.

(b) All the simple lactate- and citrate-buffer solutions ranging from pH 4.0 to pH 8.0 etched enamel, especially during the longer periods of immersion.

(c) Saliva is generally supersaturated with tricalcium phosphate, but the degree of supersaturation varies.

(d) Lactate and citrate buffers previously saturated with tricalcium phosphate etch enamel only at H-ion concentrations greater than pH 5.0.

V. BACTERIOLOGICAL SURVEY OF CARIOUS AND NON-CARIOUS PATIENTS

In 1926, after reaching the conclusion that the bacterio-chemical theory of dental caries was fundamentally sound, the bacteriological phase of our investigation was started. The published reports of four of the outstanding studies of the last decade on the bacterial flora of carious cases were in agreement as to the importance of lactobacilli in the etiology of caries. These findings of Bunting (8, 9), Rodriguez (71), McIntosh (57), and Jay (49) were also basically in accord in this respect with the results of Howe's earlier studies (43). The results obtained by these investigators agreed because they all used a strongly acid broth to prevent the growth of all except the aciduric types of microörganisms. The use of a broth adjusted to pH 5.0 practically insures the growth of a pure culture of lactobacilli from a seeding of food debris from teeth. In our experience a streptococcus yeast or oidium-like organism seldom contaminated such a culture.

In none of these bacteriological reports was there sufficient reason to justify their authors' choice of procedure, which immediately removed from consideration other acid-producing bacteria of the mouth. It seemed, before the results of our studies of the effects of acids on enamel were available, that the earlier bacteriological investigation of Kligler studying with Gies (22, 23, 24) was more adequately conceived. His technical procedure allowed consideration of the possible importance of other bacteria, such as streptococci, diplococci, and staphylococci, as well as B. acidophilus, L. buccalis, and C. placoides. He came to the conclusion that the last three mentioned were of primary importance in the causation of early caries of enamel. Kligler's conclusion concerning the bacteria involved in primary caries was limited to only three cases of this type. His study, however, merits more attention than it appears to have received, because the bacteriologic information concerning the dental zone was in a very chaotic state before his contribution in 1915.

Although Bunting's method of attack was not sufficiently broad to justify his conclusion that B. acidophilus was the active causative agent of dental caries, his findings were so consistent that it appeared he had proved that lactobacilli were always associated with decay. Therefore his method, and those of others who used a broth of pH 5.0, were chosen by us as parts of a more general procedure to determine the bacterial flora of carious enamel as compared with non-carious teeth. For the acid broth we used a galactose-whey broth, the preparation of which was described by Rettger and Cheplin in 1921 (70). Our more general procedure involved the use of blood-agar plates (pH 7.4; human blood, 7 percent). It is evident that, with the large number of types of organisms present on teeth, the types obtained will vary with differences in the media and in the prevailing environmental conditions. It seemed that the blood agar would provide, better than any other known medium, the great range of nutrients required by oral microörganisms.

A. Method of collection of specimens and of bacteriological examination

After considerable preliminary experimentation, the following method was chosen: A sterile "gold" knife and a sterile chisel-shaped

instrument were carried to the Dental Infirmary in sterile cotton-plugged test tubes. Suitable cases of early caries of the enamel and patients apparently immune to caries were selected. So much difficulty was met in finding patients with no caries that we arbitrarily defined "immune" cases as those that had no history of caries within the past five years. With the "gold" knife we scraped as much "materia alba" as possible from an incipient lesion, or from a place in the "immune" mouths where caries was most likely to begin. These specimens taken with the knife were labeled "a." Then the carious lesion was washed with cotton and sterile water, and a specimen was taken at the same site with the chisel-shaped instrument, which was designed to collect the softened ends of the enamel rods. This deep specimen was labeled "b." The bacterial types in both the "a" and "b" specimens were practically always similar, except in a few cases where the "b" specimens were sterile. Therefore, early in the investigation, we discontinued the analysis of "b" specimens. The instruments bearing the specimens, protected by the sterile test tubes, were carried to the laboratory, where examination was immediately made according to the following procedure: Two cc. of sterile distilled water were added to the tubes containing the specimens that adhered to the instruments. After each specimen had been freed into the water, the suspension, vigorously shaken until it appeared uniform, was subjected to bacteriological examination, as follows:

(a) Direct smear by the gram method.

(b) Loopful streaked several times across the surface of blood agar, and incubated at 37°C. for at least 48 hours. (If the direct smear gave an indication of the presence of the higher forms of bacteria, incubation was continued over a longer period.)

(c) The remainder of the suspension was seeded into acid galactose-whey broth (pH 5.0), and incubated at 37°C. for one week.

(d) The fermentation characteristics of each type isolated from the blood agar and acid broth were determined by inoculations into semi-solid agar or broth, or both (brom-cresol purple indicator), containing 1 percent of each of the following carbohydrates: glucose, lactose, sucrose, maltose, mannitol and inulin. Also, seedings were made in gelatin and milk. In a later study, designed to classify the lactobacilli associated with caries, characteristics of the strains were investigated on a much broader scale.

Progressive strides in the classification of bacteria have been made since the works of Miller (60, 61), Goadby (27), and Kligler (22, 23, 24) appeared. But there is still so much room for improvement, based on new information concerning variation in bacteria, that in this investigation it seemed desirable to determine under given conditions, and to record, the basic characteristics of the organisms obtained. Such a procedure would unduly lengthen this report. On this account we list, for the present, the names of the bacteria that appear, from Bergey's Manual (1), to have characteristics similar to those noted in our work. The use of certain important names is discussed in a later section of this report (page 813).

B. Results of the bacteriological survey

We had concluded the bacteriological examination of the food debris from the teeth of 148 patients before completing the study of the effects of acids on enamel. It will be seen, as this report advances, that the evidence from each of the two fundamental studies brought us to the same conclusion; namely, that the active agents of caries of the enamel are the oral microörganisms that produce and withstand H-ion concentrations equal to at least pH 5.0. Of the 148 patients, 85 had incipient caries and 63 were "immune" on the first clinical examination when the bacteriological specimens were collected. An attempted correlation of the findings of the bacteriological with the clinical examinations gave only confusing information, owing chiefly to the fact that diagnosis from only one clinical and one bacteriological examination may be very misleading. This discovery was made after 50 of the 148 patients had been subjected to the follow-up examinations at from three to ten months succeeding the first diagnosis. The repetition of clinical examinations often showed that what at the first examination had been regarded as progressive caries was actually not this type at all. Many of the "immune" cases showed, on subsequent examinations, that they were affected with progressive caries of the enamel. Even when attention was concentrated on the relation of the lactobacilli isolated by acid broth to the clinical diagnosis of one examination, the same misleading information was manifest. Of the 85 patients exhibiting incipient caries, the cultures for 53, or 61 percent, had lactobacilli; of the 63 patients free from caries, the cultures for 33, or 52 percent, also showed the presence of lactobacilli. This

evidence certainly does not indicate that lactobacilli are important factors in the etiology of caries of the enamel, but neither does it contraindicate that importance to one who considers the conditions that exist, if the bacterio-chemical theory of tooth decay is true. If acid from fermented carbohydrates causes dental caries, it is obvious that there are periods when the bacteria conducive to caries are present, but have as yet produced no lesion observable macroscopically or with the aid of an explorer. There also might be lesions which to all outward appearances are active, but have lost the harmful microörganisms. Therefore, single clinical and bacteriological examinations in given cases might yield the contradictory results referred to above.

A system of follow-up examinations seems to be the dependable method of determining whether caries is active or arrested, and whether certain microörganisms are significant in the carious process. This fact is clearly demonstrated by the results for the 50 patients who were subjected to follow-up examinations. If we take into account only the evidence from the first examination, there was no suggestion that lactobacilli are important factors. Of these 50 cases, 35 were recorded as non-carious, of which 18 were positive for lactobacilli and 17 negative. Of the 15 carious cases, 9 were positive for lactobacilli and 6 negative. Upon making follow-up examinations three to nine months after the first, it was found that the 6 cases of incipient caries without lactobacilli had not advanced; of the 9 cases of incipient caries with lactobacilli present on the first examination, 5 had become advanced caries. We have here strong circumstantial evidence of the importance of lactobacilli in the carious process. On further consideration of the 35 non-carious cases, it was found that 13 of the 18 cases that were positive for lactobacilli on the first examination had developed caries by the time of the second examination three to four months later, whereas 16 of the 17 cases that were negative for lactobacilli on the first examination were still without caries on the second examination. From the evidence thus gathered the inference was made, in 1927, that the initial bacteriological findings with respect to the presence or absence of lactobacilli seemed to be a true prognostic indicator of the conditions that would be observed on clinical examination three to six months later. Jay (49) came to this conclusion independently, as is evidenced in his report of 1929.

The data in *table 5* relate only to the 50 patients who were subjected to the follow-up examinations. As this general bacteriological phase of our work was not extensive, the evidence cannot be convincing. But the general agreement of the results of our study of the effects of acids on enamel with those of our general bacteriological investigations,

TABLE 5

*Number of times different microörganisms were present in bacterial plaques in carious and non-carious mouths**

BACTERIAL TYPES	CASES OF PROGRESSIVE CARIES (19)		CASES OF NEGATIVE OR NON-PROGRESSIVE CARIES (31)	
	No. of times found	Percent	No. of times found	Percent
M. conglomeratus	4		3	
M. ochræus	3		2	
C. pseudodiphtericum	6		13	
N. sicca	3		5	
Str. mitior	16	84	29	94
L. acidophilus (Y type)	18	95	7	23
G. crassus	3		6	
Pseudomonas	2		1	
L. buccalis	17	90	23	74
B. mesentericus	2		3	
N. flavus	9	47	9	26
B. fusiformis	1		1	
S. albus	2		4	
N. perflavus	10	53	17	55
N. catarrhalis	1		2	
Cl. placoides	0		4	
H. influenzae	0		2	

* In the 98 patients which were not subjected to follow-up examinations, D. pneumoniae, A. recti, M. subgranulatus, B. vincenti, S. aureus, and L. acidophilus (X type) were occasionally found. This indicates that our general method of bacteriological study allowed us to find twenty-three different types of bacteria according to present standards of classification.

supplementing the similar results of Bunting, Rodriguez, Jay and MacIntosh, prompted us to defer further bacteriological and clinical study until a satisfactory antiseptic could be found that would allow us to kill, or at least to diminish appreciably at will, the lactobacilli in food debris on the teeth of patients suffering from caries. Carefully controlled clinical tests are now being carried on to determine whether a

reduction of the lactobacillus infection of the dental zone is accompanied by a decrease in the incidence of new caries. These tests are not yet complete, but have already given results that confirm the importance of the lactobacilli in the causation of tooth decay. This work will be reported in a later communication.

As to the interpretation of the evidence recorded in *table 5*, there is a definite suggestion that L. acidophilus (Y type) is the most important organism associated with progressive caries of the enamel. The general bacteriological study of the food debris on teeth has also suggested that perhaps the natural association of the lactobacilli with Leptothrix buccalis, N. flavus, and N. perflavus, might be conducive to caries. At present the information on this point is quite vague. But we have seen that these microörganisms, because of the characteristic growth of their colonies, may be important parts in plaques, which effectively hold the buffering, neutralizing, and dilutory powers of the mixed saliva at a minimum in the immediate vicinity of the carious process.

C. The nature of plaques

The related factor in the carious process that confines the action of fermentation acids to enamel, and prevents their dilution or neutralization by the general oral secretions, has received wide consideration by all investigators of dental caries who believed in the bacterio-chemical theory. Thus far it appears that only Williams (81), who first applied the term "plaque," has made important progress in this phase of research. He developed such a refined technic that he was able to demonstrate on the surface of carious lesions colonies of bacteria mixed with food debris and mucus in situ in ground sections. The varieties of terminology to define plaques such as "bacterial, gelatinoid, mucinous, and acid-mucin plaque" that have appeared in the literature might lead one to believe that some specific information concerning plaques had been contributed. However, the reports on the nature of plaques, with but few exceptions, appear to be speculative.

There is a current belief that the plaque is formed by the deposit of *acid mucin* and that Gies was responsible for this conception. An exhaustive study of Gies' reports (21, 22, 23, 24, 25) has not yet revealed that he believed that the plaques are formed of *acid mucin*. However, his discussion is the most specific and suggestive found to

date. He believes that the relatively large colony of Cladothrix placoides, which adheres tenaciously to smooth surfaces, may be an important factor in plaque *formation*. These large colonies tend to enclose other colonies of bacteria, viscid *mucinate*, delicate *mucous* threads, and food debris, in such a way that acid effect is localized on the enamel. He notes that the term "mucin plaque" is used conveniently to refer to any *mucinous* mass on the teeth, and says: "The remarks in which this phrase is used are not intended to suggest a belief that dental caries occurs only through the mechanical intervention of such mucinous masses."

The nature of the plaque has been so puzzling that our study of this subject, which is still under way, has thus far failed to add any important new knowledge to this subject. The recently acquired knowledge of bacterial variations might favor a more effective plan of attack on the nature of the filamentous forms of oral bacteria, and also facilitate a solution of complex problems in this field.

VI. NOMENCLATORIAL STUDY OF LACTOBACILLI ASSOCIATED WITH DENTAL CARIES

A. Historical

The fact that several workers have found lactobacilli in association with caries of enamel has raised the question: What is the species of these lactobacilli? This question is chiefly bacteriologic in nature, as the common tooth strains found by Morishita (62, 63), Rodriguez (71), and Bunting (8, 9, 10, 11, 12) agree in general characteristics with the tooth strain most commonly found in our studies. Whether these tooth strains should be named Lactobacillus acidophilus, odontolyticus, or acidophilus odontolyticus, the fact remains that recent investigators of this subject have been referring to organisms that are nearly if not wholly alike in morphological, biochemical, and serological characteristics. The supplementary evidence in preceding sections, indicating the importance of these oral lactobacilli in the causation of decay of tooth enamel, made it advisable to undertake taxonomic studies of oral lactobacilli for the purpose of selecting the best name for these tooth strains.

Three names appear to be available for this species of lactobacillus. Bunting and his co-workers believe that *Lactobacillus acidophilus* is

the correct name. Morishita thinks that *Lactobacillus odontolyticus*, as suggested by Rodriguez in 1921, is a more desirable name. McIntosh, James, and Lazarus-Barlow called the tooth strain *Lactobacillus acidophilus odontolyticus*. The selection of a name that will cause the least confusion depends chiefly on the correct answer to the question: Is the oral species of lactobacillus identical with the intestinal species named acidophilus by Moro (64) in 1900? This answer has been sought within the last few years by Morishita (62, 63), Rosebury, Linton, and Buchbinder (72), Howitt (47), and Hadley, Bunting and Delves (30). In the earlier paper of Morishita (62), he reported that "the tooth organisms discussed here constitute a type entirely separate from B. acidophilus of the intestinal tract;" in his last paper he reported that "it is impossible to say definitely whether the different organisms described by various workers are entirely distinct species, or whether they are different forms of one or the same species." Rosebury (72) and his associates expressed the opinion that the cultural differences between oral and intestinal strains also existed between the intestinal strains as a group, and that there was therefore no justification in separating these organisms into distinct groups. Hadley, Bunting, and Delves (30), believing that the tooth strains are identical with the intestinal strains, maintain that Lactobacillus acidophilus should be the chosen name. Rodriguez (71) and McIntosh (57), by choosing the names L. odontolyticus and L. acidophilus odontolyticus, respectively, early indicated their belief that there were essential differences between the oral and intestinal strains. Still earlier, Kligler (22, 23, 24) and Howe (43) used the name B. acidophilus, although the latter found many divergent types which he termed B. bifidus, and Bacillus X, Y and M.

In our broad taxonomic study of this problem, thirty tooth strains, isolated by us from early caries of the enamel, were compared with several known intestinal strains as well as with many other species of the Lactobacillus genus. Rather than unduly unbalance this contribution on the cause and nature of dental caries with a lengthy report on the classification of lactobacilli generally, this section will be confined to statements on dental strains and their relation to intestinal strains of lactobacilli.

B. Procedure for nomenclatorial study

Our first step in the identification of dental strains of lactobacilli was the isolation and selection of strains from scrapings of early caries of the enamel. This work was accomplished by seedings into galactose-whey broth (pH 5.0), and later by repeated platings of isolated lactobacilli colonies in galactose whey or tomato agar. Thirty strains were selected on the basis that they were probably pure strains, as evidenced by repeated examinations of smears and colonial characteristics in alternate cultures in broth and agar. The second step was to learn the fermentive characteristics of these thirty strains on fourteen different carbohydrates or alcohols. The results of this phase of our study allowed us to choose a strain (114) that was similar in its characteristics to so many other strains that it could rightfully be labeled "our commonly found dental strain." The third step was to compare this # 114 strain with strains that had been commonly found by Rodriguez, Bunting, and Morishita. When it was learned that these dental strains, found by four different students of dental caries in four different cities, were identical, we proceeded to the next step, which was to compare these thirty-three dental strains with six intestinal strains of B. acidophilus. After a broad comparative study of the dental and intestinal strains had indicated that the commonly found dental types could be isolated from certain commercial "acidophilus preparations," and that five of the thirty dental strains simulated very closely the rough or fuzzy type of intestinal acidophilus, we sought to determine whether the different main types found in both sources could be dissociated.

Of the six intestinal strains used in this investigation, three—*Scav.*, *R-1-1*, and *Pack*—were obtained from the Department of Bacteriology at Yale University through the courtesy of Dr. L. F. Rettger. The other three strains were isolated in our laboratory from three different commercial acidophilus preparations: *DM* from a solid preparation, *DL* from an acidophilus milk, both of which were sold to consumers for immediate consumption; *BK* from an acidophilus-milk seeding-culture distributed from a commercial laboratory to large dairies for seeding purposes.

C. Morphological study

The main intensive study of the morphological characteristics of the thirty-three dental and six intestinal strains was made in Gram smears of 1-, 2-, 3-, and 7-day cultures in two different media—casein-digest broth, and milk. The immediate sources of the strains were stock milk-cultures that had been transferred bi-monthly for over a year before we began this phase of the investigation. For several days before obtaining smears for the Gram stain, daily transfers were made in the broth and milk of all thirty-nine strains to assure ourselves that all were actively growing cultures. The same supplies of neutral broth and milk were used during the entire comparative study. All Gram stains were made with the technic that had previously been found satisfactory.

In this study all the forms that have been described in detail by Howe, Rodriguez, Bunting, Morishita, and Rosebury have been seen in both the intestinal and dental groups. We have found variable forms in many cultures, not only in different cultures, but also in the same culture at different periods of growth. In broth cultures more than in milk there has been a definite trend of early-culture rods to older-culture coccoid forms. It appears that no constructive classificational use can be made of our available knowledge of morphology. In *tables 6*, *8*, and *9*, only the young-culture shape is given, and only a superficial description of that. It seems that the most important information gained from the morphological study is that both intestinal and dental strains such as Scav., R-1-1, Pack, D144 and 160 could be differentiated from strains 114 or B K, in that the former tended to be more filamentous. We believe that other morphological characteristics could also be used to differentiate these two main types, but studies of a biochemical and serological nature have indicated that these tests could be used to better advantage for this purpose.

D. Fermentation study

The fermentation of the carbohydrates, alcohols, and glucoside noted in *tables 6*, *8*, and *9* was determined on several occasions for the entire series of thirty strains, using either sugar-free casein-digest broth or semi-solid agar as the medium. The test substances were

sterilized in neutral water solutions by heat in all cases excepting levulose, maltose, and sucrose, which were subjected to filtration through Mandler filters. The test media contained brom-cresol purple and 1 percent of the test substance. The attainment of pH 6.0, or lower, within an incubation period of seven days at 37°C., was considered positive. The test substances, arabinose and xylose, often were fermented close to pH 6.0, but only in isolated instances beyond that point. The + and − signs in the tables indicate acid production or none, respectively; + has been reported only when at least two-thirds of the three to twelve series of tests on each of the test substances gave acid production to pH 6.0 or lower. Where ± is noted, + or − was observed in about 50 percent of the several tests made. After three comparative series of tests in which casein-digest semi-solid agar and broth were employed, the use of the agar medium was discontinued, because false negatives appeared in such an environment more often than in the broth.

For seeding material, actively growing plain casein-digest broth-cultures, obtained from daily transfers, were used if growth was luxuriant enough. In certain slow growing cultures glucose was added to the seeding cultures, and, after growth was heavy, the cultures were centrifuged aseptically. Next, the washed sediment was suspended in physiological saline for seeding material. Other precautions taken in this fermentation study were the use of extra heavy seedings in all sugar-broth cultures showing negative acid production in the regular tests; and also the use of a Shiga strain of dysentery in all supplies of culture material containing fermentable substances, to make certain that no dextrose resulting from the sterilizing procedures was present.

An analysis of the fermentation results in *table 6*, and of the data in *table 7*, indicated that it was advisable, for purposes of exposition, to classify our thirty strains into four groups, as follows:

Group I. Strains that produced acid and no gas in dextrose, levulose, galactose, maltose, lactose, sucrose, mannitol, sorbitol, salicin, and dextrin; they produced *neither* acid nor gas in arabinose, xylose, raffinose, and inulin: 55, 64, 107, 110, 114, 157, 162, D 147, D 158, D 164, D 165, D 168, D 155. Strains 45, 167, 172, and D 171 are also placed in this group because they differed only in the fact that they failed to ferment sucrose in two-thirds of the tests made on this sugar.

TABLE 6

*Characteristics of the dental strains**

DENTAL STRAINS	MORPHOLOGY				COLONY FORM				ACID PRODUCTION IN DIFFERENT MEDIA													LIMITING pH IN WHEY BROTH	GREATEST DILUTIONS OF NO. 114 ANTI-SERUM GIVEN + AGGLUT.
	Long rods	Short rods	Coccoid	Beaded	X	Y	Xy	Yx	Dextrose	Levulose	Galactose	Arabinose	Xylose	Maltose	Lactose	Sucrose	Raffinose	Mannitol	Sorbitol	Salicin	Dextrin	Inulin	
45	+		+			+			+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.8	1:320
55	+	+				+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.9	1:320
64	+	+						+	+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:320
107	+	+						+	+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:320
110	+						+		+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.9	1:320
114	+							+	+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.9	1:1280
157	+					+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.9	1:640
160	+		+	+	+				+	+	+	−	−	+	+	+	+	−	−	−	±	−	4.2	None
161	+	+						+	+	+	+	−	−	−	+	−	−	−	+	−	−	−		None
162	+					+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:640
167	+					+			+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.9	1:640
172		+							+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.6	None
174	+	+	+			+			+	+	+	−	−	+	+	+	+	+	+	+	+	−		None
175	+		+			+			+	+	+	−	−	+	+	+	+	+	+	+	+	−		None
D144		+			+				+	+	+	−	−	+	+	+	−	−	−	−	±	−	4.0	None
D147		+				+			+	+	+	−	−	+	±	+	−	+	+	+	+	−	3.4	1:160
D149	+		+		+				+	+	+	−	−	+	+	±	−	±	±	±	±	−	3.4	None
D150	+		+	+					+	+	+	−	−	+	±	±	−	−	−	−	−	−		None
D152	+			+			+		+	+	+	−	−	+	+	+	+	−	−	−	−	−		None
D156	+	+							+	+	+	−	−	+	+	+	+	−	−	−	−	−	4.4	None
D158	+	+						+	+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:1280
D159									+	+	+	−	−	+	+	+	−	+	+	−	−	−		None
D164	+					+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	4.0	1:1280
D165	+					+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	4.0	1:1280
D167	+					+			+	+	+	−	−	+	+	+	+	−	−	−	±	−	4.0	None
D168	+	+				+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:320
D170	+	+						+	+	+	+	−	−	+	+	−	+	−	−	−	±	−	4.5	None
D171	+		+			+			+	+	+	−	−	+	+	−	−	+	+	+	+	−		None
D155	+		+					+	+	+	+	−	−	+	+	+	−	+	+	+	+	−		None
D163	+		+					+	+	+	+	−	−	+	+	+	+	+	+	+	+	−		None

* Explanation of the symbols: X = rough type of colony. Y = smooth type of colony. Xy = predominantly X type. Yx = predominantly Y type.

Group II. Strains that differed from the fermentive activities of Group I in not producing acid in sorbitol, mannitol, salicin, or dextrin, or questionably in the latter; they produced acid in raffinose: 160, D 144, D 152, D 156, D 167.

TABLE 7

Analysis of the data of fermentation results in table 6

GROUP	FERMENTATION CHARACTERISTICS	NUMBER OF STRAINS
I	*Acid and no gas* in dextrose, levulose, galactose, maltose, lactose, sucrose, mannitol, sorbitol, salicin, and dextrin. *Neither acid nor gas* in arabinose, xylose, raffinose, and inulin.	17
II	Same as Group I, excepting that *neither acid nor gas* was produced in sorbitol, mannitol, salicin, or dextrin, or questionably in the latter. All these strains *produced acid* in raffinose.	5
III	Showed all the positive fermentive activities of Groups I and II. Arabinose, xylose, and inulin were the only carbohydrates *not fermented.*	3
IV	Heterogeneous strains, each of which varied in a different manner, which may be determined from *table 6.*	5

TABLE 8

Comparison of dental strains commonly found by different investigators

DENTAL STRAINS COMMONLY FOUND BY SEVERAL INVESTIGATORS	MORPHOLOGY				COLONY FORM				PRODUCTION OF ACID IN													LIMITING pH IN WHEY BROTH	GREATEST DILUTION OF NO. 114 ANTISERUM GIVEN + AGGLUT.
	Long rods	Short rods	Coccoid	Beaded	X	Y	Xy	Yx	Dextrose	Levulose	Galactose	Arabinose	Xylose	Maltose	Lactose	Sucrose	Raffinose	Mannitol	Sorbitol	Salicin	Dextrin	Inulin	
Bunting....	+					+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	4.0	1:1280
Rodriguez..	+	+				+			+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.8	1:1280
Morishita...	+	+				+			+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.8	1:640
Enright.....	+	+				+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.9	1:1280

TABLE 9

Characteristics of intestinal strains

INTESTINAL STRAINS	MORPHOLOGY				COLONY FORM				PRODUCTION OF ACID IN													LIMITING pH IN WHEY BROTH	GREATEST DILUTION OF NO. 114 ANTISERUM GIVEN + AGGLUT.
	Long rods	Short rods	Coccoid	Beaded	X	Y	Xy	Yx	Dextrose	Levulose	Galactose	Arabinose	Xylose	Maltose	Lactose	Sucrose	Raffinose	Mannitol	Sorbitol	Salicin	Dextrin	Inulin	
Scav.....	+			+	+				+	+	+	−	−	+	+	−	+	−	−	−	−	−	4.4	None
R–1–1	+			+	+				+	+	+	−	−	+	+	+	+	−	−	−	−	−	4.4	None
Pack.....	+			+	+				+	+	+	−	−	+	+	+	+	−	−	−	−	−	4.8	None
DM.....	+	+				+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.8	1:640
DL.....		+						+	+	+	+	−	−	+	+	−	−	+	+	+	+	−	3.9	1:1280
BK.....	+	+				+			+	+	+	−	−	+	+	+	−	+	+	+	+	−	3.6	1:1280

Group III. Strains that produced acid in all the test substances except arabinose, xylose, and inulin: 174, 175, D 163. The strains of this group appear to be mixtures of the two types previously classified in Groups I and II.

Group IV. Miscellaneous strains; dissimilar; see *table 6*: 161, D 149, D 150, D 159, D 170.

From this classification of the thirty dental strains intensively studied, according to fermentation characteristics, Group I is obviously our most commonly found type. The next step was to learn whether our commonly found dental strains behaved fermentively like the most commonly found dental strains of Bunting, Rodriguez, and Morishita. The results of this phase of our study (*table 8*) showed that the strains (which we received labeled as those most commonly found by these investigators) all belonged to the important Group I. We were then ready for the important task of comparing the fermentive activities of the dental lactobacilli with those of the intestinal type. A study of the results in *table 9* shows that the Y-type intestinal strains behave like the Group-I dental strains, and that the X-type intestinal strains behave like the Group II dental strains.

E. Agglutination study

A relatively broad study of the serological relationships of the different strains has been completed only to the point where the agglutinability of the organisms has been determined with respect to common tooth-strain 114 and Y intestinal-strain BK-antisera. It is unfortunate that rabbits which were being injected with vaccines of Group II-type dental-strain 160 and intestinal-strain Scav. died before the antisera of these types were procured. It is probably coincidental that all four rabbits receiving injections of raffinose-positive and mannitol-negative fermenters died, because two of the rabbits receiving injections of the raffinose-negative and mannitol-positive also died. The animals from which we obtained the 114 and BK antisera showed no signs of illness, and gradually increased in weight during the period of antisera production.

The agglutination results in *tables 6*, *8*, and *9* are only those given by common tooth-strain 114, because the results obtained with intestinal-strain BK were practically identical. A comparative study of

the fermentation and agglutination results in these tables shows that a remarkably consistent correlation has been found. Of the seventeen dental strains belonging to Group I, fourteen were agglutinated by the common tooth-strain antiserum as also were all three Group-I intestinal strains. There was no cross agglutination of any strain having fermentation reactions different from those exhibited by the organism of Group-I type. The strains that were agglutinated by our common tooth-strain and by intestinal-strain BK antisera were 45, 55, 64, 107, 110, 114, 157, 162, 167, D 147, D 158, D 164, D 165, and D 168.

F. Growth at low temperature

Morishita (62) reported that one of the most important means of separating the intestinal from the dental type of lactobacilli was based on the fact that the latter were unable to grow at temperatures as low as 12–14°C., while the former failed to do so even at 18°C. Experiments were carried out to test the general validity of this statement by incubating our dental and intestinal strains at 15°C. and 37°C., after seeding heavily in neutral tomato-broth containing an addition of 1 percent of glucose. The results of the tests, which were allowed to run twelve days, are recorded in *table 10*.

The results of growth at 15°C. by the various strains, as shown in *table 10*, are very interesting. All the lactobacilli of both dental and intestinal origins in Group I, according to fermentive and agglutination results, grew at 15°C. On the other hand, of the six strains that did not grow at 15°C., three of the intestinal and two of the dental belong to Group II. The other strain, 175, although placed in Group III in accordance with most of the fermentive findings, acted like a Group-II strain during our tests of growth at 15°C.

G. Summary

To summarize briefly the results of these studies of lactobacilli of dental and intestinal origins, the evidence indicates that there are two main types that may be differentiated quite easily by a combination of morphological, fermentation, agglutination, and growth (at 15°C.) characteristics. The Group-I type usually produces a smooth colony, and acid without gas in glucose, galactose, levulose, maltose, lactose, sucrose, mannitol, sorbitol, dextrin, and salicin; does not produce acid

or gas in raffinose, arabinose, xylose, or inulin. The strains of this type grow at 15°C., and are agglutinated by an antiserum produced by injection of suspensions of a Group-I organism into a rabbit. The Group-II type produces usually a rough fuzzy type of colony, and acid

TABLE 10

Effects of different temperatures on the growth of lactobacilli, of so-called dental and intestinal origin, in tomato broth

DENTAL STRAINS	45	55	64	107	110	114	157	160	161	162	167	172	Rod.
Number of days before definite growth occurred at													
37°C.	1	1	1	1	1	1	1	1	5	1	5	1	1
15°C.	5	5	5	5	5	5	5	None in 12	2	3	5	5	5

DENTAL STRAINS	175	144	D147	D149	D152	D156	D158	D164	D165	D168	D170	BN	Mor.
Number of days before definite growth occurred at													
37°C.	1	1	1	1	2	1	2	1	1	1	1	1	1
15°C.	None in 12	None in 12	8	5	5	8	5	5	5	5	12	5	5

INTESTINAL STRAINS	Scav.	R-1-1	Pack	DM	DL	BK
Number of days before definite growth occurred at						
37°C.	2	2	1	1	1	1
15°C.	None in 12	None in 12	None in 12	12	3	5

in glucose, galactose, levulose, maltose, lactose, sucrose, and raffinose; may or may not produce acid in dextrin; usually does not produce acid or gas in arabinose, xylose, mannitol, sorbitol, salicin, or inulin. The strains of this type do not grow at 15°C., nor are they agglutinated by Group-I antiserum. Each of the two main types is found among strains of dental and intestinal origin.

H. Dissociation study

The question now arises as to whether these two types are different phases in the life cycles of the same organism. There have been many clues indicating that such is the case. Thus, when strain DL was isolated from acidophilus milk intended for intestinal therapy in 1926, it was a fuzzy rough colony, which we have learned from an authentic source simulated very closely the Scav. strain when it was seeded into the commercial milk-preparation. We carried it on a bi-monthly transfer for two years in milk before we started to study the strain intensively. During this intensive study of the strain, DL changed into a form that very often gave pure cultures of smooth colonies, but became a Group I type in all other characteristics. Another observation indicating the likelihood that the X- and Y-types are variants of the same organism is that reported by Kulp and Rettger (51), who said that, in their comparative studies of L. acidophilus and L. bulgaricus—although all the primary isolations labeled L. acidophilus were made from the extreme X-type colonies—the Y-types appeared quite often in subsequent platings.

Although these two random observations might be augmented by many others of the same significance, it will be sufficient to allude to the more recent observations of Hadley, Bunting, and Delves (30), who reported a systematic study of the variability of individual strains of L. acidophilus, which they dissociated by cultivation from the rough filamentous borders or marginal outgrowths that occur under certain conditions on the common smooth type of colony. These workers found that, by simple laboratory procedures, they were able to transform "smooth" oral strains into "rough" ones, and rough intestinal strains into smooth ones. These findings, if confirmed, have a very important bearing on the proper name of the lactobacilli associated with dental caries. Although details as to how dissociation was effected are lacking in their paper, we have attempted on a small scale to change our commonly found tooth-strain 114 from a smooth to a rough type, and also the rough intestinal strain Scav. into a smooth type, by transfers alternatively from broth to agar and agar to broth, varying the period of incubation in each medium from eighteen hours to several days. Thus far agar-plate colonies have been observed after different seedings. On one occasion we saw marginal outgrowths of a

rough type from the smooth 114 colony, but transfers from this area gave a typical diffuse turbid growth in broth, and the subsequent platings in agar showed only regular smooth colonies. No success in dissociating the rough Scav. type has been had during our relatively brief study of this phenomenon.

Conclusion

The evidence in this chapter supports the view that the lactobacilli (Group I of *tables 6, 7, 8*) associated with the causation of caries of the enamel are L. acidophilus, Y type.

VII. SUMMARY AND CONCLUSIONS

1. An extensive study of the literature and of our own results indicates that (*a*) local environmental conditions are the main factors in the *active* causation of decay of fully erupted enamel, and that (*b*) deficient diet and defective nutrition, by being responsible for the construction of teeth more susceptible to the action of the acids of fermentation, are the most important factors during the period of development of the teeth. These studies also indicate that improper diet and defective nutrition, by being responsible for a salivary environment low in content of calcium phosphate, are of some, though minor, importance as a predisposing factor in caries of enamel.

2. Artificial lesions that simulate natural caries of the enamel have been prepared.

3. The halt that is sometimes observed in carious processes of the enamel is regarded as being due to the presence of acid-insoluble protective organic coatings. Demonstrations of this phenomenon are offered.

4. Explanation is given of certain shortcomings in the views of Miller's followers, such as the reason why mouths in filthy hygienic condition are often free from caries, and why carious lesions may increase, and make more rapid progress, during pregnancy.

5. Simple lactate- and citrate-buffer solutions ranging in H-ion concentration from pH 4.0 to pH 8.0 dissolved tooth enamel in the longer immersion periods.

6. Different tooth enamels vary in their susceptibility to the action of acids.

7. Human saliva is generally saturated with tricalcium phosphate.

8. Lactate- and citrate-buffer solutions previously saturated with tricalcium phosphate dissolve enamel only at H-ion concentrations greater than approximately pH 5.0.

9. The only microörganism commonly found in the food debris, in direct contact with progressive caries of the enamel, that can tolerate and produce additional acid in an environment below pH 5.0, is a lactobacillus having the following characteristics: gram-positive rod, which usually produces (*a*) a smooth colony; (*b*) acid but no gas from dextrose, levulose, galactose, maltose, lactose, sucrose, mannitol, sorbitol, salicin, and dextrin; (*c*) neither acid nor gas from arabinose, xylose, raffinose, and inulin. Its final pH in glucose-tomato broth generally ranges from 3.6 to 4.0. It can grow at a temperature of 15°C. It is agglutinated in high dilution by the Y type of L. acidophilus antiserum, which does not at all agglutinate L. acidophilus of the X type.

10. Carefully controlled clinical tests are now being conducted to determine whether a reduction in the lactobacillus infection of the dental zone is accompanied by a decrease in the incidence of new caries. These tests, which are now in the second year, are already giving results that confirm the importance of the lactobacilli in the causation of tooth decay. This work will be reported in a later communication.

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IX. DISCUSSION

William J. Gies, Ph.D., F.A.C.D. (New York City): [The (a) public, (b) dental profession, (c) Pittsburgh Section of the Research Association, (d) audience, and (e) authors were congratulated, respectively, on (a) the public benefits steadily accruing from the advancement of dental science and dental practice, (b) the earnest endeavors of the dental profession to meet its public responsibility by promoting this advancement, (c) the constructive activity of the Pittsburgh Section, (d) the opportunity of the large audience to receive the results of a prolonged and earnest research, (e) and the effective way in which a solution of the problem of dental caries was sought and investigated. Like cancer and colds in the practice of medicine, caries in the practice of dentistry remains a curse and a puzzle requiring and receiving intensive attention for its solution. Current views on the origin and prevention of caries were given general attention not only to point out the complex difficulties the authors have been obliged to face, but also to prepare the way for the more detailed and technical discussions by the succeeding speakers.]

Heredity presumably has some relation to the problem—how much, is not clear. It is often said, in relation to prevention, that one should be very wise in the selection of his great-grandparents. In recent years there has been a tendency to exaggerate and misrepresent dietary relationships. No one knows what particular diet or diets, if any, will insure the development of normal teeth and prevent dental caries. When the dietary findings of many investigators are compared, we see that, despite the earnestness and efficiency of their efforts, much remains uncertain. Thus, Hawkins has been mentioned as one of those who have published important findings on dietary relationships. Hawkins ascribes preventive results to conditions of diet that include *low* content of cereal, *medium* content of protein, *high* content of calcium, and *high* potential alkalinity. The following quotation will interest you not only as directly disagreeing with Hawkins's *explanations* of his results, but also as indicating the need for further clarification of the recorded observations in this important field.

"The hygienic condition of certain of the inhabitants of the Island of Lewis in the Hebrides could scarcely be worse. They live in 'black houses,' the walls of which are often as much as five feet thick. There is no chimney and only one opening in the house. Peat fires are kept constantly burning. Domestic animals occupy the building with the family. Chickens roost on the beds, tables, and chairs. The children are rarely ever taken out of the house until they can walk, yet rickets is almost unknown on the island; and .... 'the most striking fact in the adult population [of course, that includes their childhood also] is their beautiful teeth—a testimony to the absence of rickets in infancy.'

Furthermore the infant death-rate is among the lowest in the British Isles. . . . . The staple articles of food are fish, oatmeal, and eggs. Milk is scarce and vegetables almost unknown. Fish liver is a favorite dish. Such a diet is *high* in protein and phosphorus, and *low* in calcium and potassium. It has a relatively high potential *acidity*, and is probably also rich in vitamins. Such a combination of dietary factors apparently is able to overcome the deleterious effects of poor hygiene, filth, and lack of ultraviolet radiation."[10] [Italic not in the original.]

The four dietary factors to which Hawkins ascribes special preventive virtue are *moderate* protein, *low* cereal, *high* calcium, and high potential *alkalinity*. But the diet common among the people on the Island of Lewis, as described in the above quotation, disagrees in quality with each of Hawkins' chemical criteria; that is to say, their diet is *high* in protein, *high* in cereal, *low* in calcium, and high in potential *acidity*. The conditions on the Island of Lewis to which the above quotation alludes have been operative there for many years, and the quoted observations refer to a larger number of people through a longer period of time than for either in Hawkins' experiments. In these two comparative instances, two sets of dietary factors, representing opposites in nutritional quality, are reported as having had exceptionally beneficial effects on teeth. This striking disagreement in scientific *explanations* justifies the opinion that errors have been made in arriving at the reported conclusions, in one case or the other, or both; or that essential factors, which were overlooked or disregarded in the observations, have had important favoring influences. Evidently something is lacking in our present understanding of the kinds of diet to prescribe for the production and maintenance of perfect teeth, if these conditions are attainable by dietary influences alone.

Some earnest investigators believe that protection against dental decay comes wholly or primarily from within the tooth, and that the way to prevent dental decay is to improve the general health of the person. There are varieties of this theory, but all involve the assumption that the enamel is a *source* of vital protective activity, or is in such an intimate relation with vital changes nearby, in the dentin and pulp, that the enamel receives the benefit of actively protective vital influences by way of these tissues. One of the leading observers in this relation is Fish, of London. He has made numerous experiments on the permeability of enamel, and the relation of its permeability to the dentin and pulp. His work is often cited as a basis for the conclusion that the enamel is actively vital, and that decay is normally prevented from the inside of the tooth. Fish, after further investigation, has lately changed his opinion. This is evidence of an open mind, but also evidence of the difficulties confronting the earnest student and investigator

[10] Gies: *Journal of Dental Research*, 1931, xi, p. 221; April.

who tries to find the real reasons for dental decay. I quote the general conclusions in a recent publication by Fish—they afford opportunity to make direct comparisons with the work reported by Drs. Friesell and Enright:

"1. Caries appears to be produced as a result of fermentation and proteolysis in permeable faults of the enamel.

"2. The faults consist of permeable lamellae, and soft white patches of enamel on the sides of the teeth and in the molar fissures. They are hereditary, being very numerous in the teeth of the members of some families and rare in others. Teeth which are free from these faults are free from caries (except the cervical variety).

"3. These faults are not the result of vitamin deficiency, because (*a*) they are not distributed at points corresponding to any particular stage of development of the teeth; (*b*) the comparative freedom of certain families, one generation after another, from these faults, indicates that they are definitely inherited; (*c*) vitamin D is plentiful in children's food, and the well-to-do children are no more immune than the poor; and (*d*) it is inconceivable that 85 per cent or more of the population suffer from vitamin-D deficiency without any other symptoms than hypoplasia of the enamel.

"4. The mouth, as an environment for the microörganisms producing caries in these faults, is an important factor in etiology. The mouth may be completely immune, as in the dog, or may be partially immune, as in certain human beings with arrested caries; or susceptible, as in the case of ordinary caries.

"5. There is little possibility of an actual resistance being offered to the spread of caries in the tooth by the dentine under the lesion because it is dead, but the surrounding live dentine can hypercalcify, and so hold up the progress of the disease.

"6. The Streptococcus mutans appears in pure culture in the dead tract of tubules under caries, and appears to be the causative organism."[11]

When I intimate that Drs. Friesell and Enright encountered serious difficulties in their research, you will understand from the two quotations I have read that if any one should disagree with them, that fact would not imply more reliable information than theirs. We are all groping toward a solution. Every effective effort contributes something important to the ultimate clarification. Sooner or later the gaps will be filled, and the problem solved.

The papers presented tonight—in their discussions of the clinical realities, of the action of acids on enamel, and of the bacteriological data—are very long steps forward. Several general questions arise. Assuming that saliva can be made to exercise influences that are highly protective—that is to say, can be given the concentration of calcium phosphates that will protect the teeth against the acids produced by bacteria—will that protection apply to any of the defects, to any of the pits and fissures, *where the saliva ordinarily does not arrive?* Will the protection apply to those conformations of the enamel that are not freely bathed by saliva? Will it apply to those conformations of the enamel where plaques, particles of food, etc., are apt to be

[11] Fish: *Dental Surgeon* (London), 1932, xxix, p. 271; May 14.

held and there interfere with the movement of saliva, besides favoring destructive acid production "on the spot." In other words, does saliva, however highly protective it may be, ordinarily gain access often enough, or abundantly enough, in various "susceptible" areas *there* to perform a protective function? If the answer to this question is "No," an additional question arises: How could such saliva, *or its equivalent*, be *made* to protect these "susceptible" defects and conformations?

Russell W. Bunting, D.D.Sc., F.A.C.D. (Ann Arbor, Michigan): Those of us who have been invited to take part in this discussion have been having a fine time today in a conference over questions of mutual interest, most of us being primarily interested in a study of dental caries, some having it as our major interest, and others having general information on the subject. The curious thing about this conference was that as we talked over the various controversial and mooted questions, we found many points of agreement. I believe some of us thought we were going to have a jolly scrap. Some people question whether investigators can agree on anything, but we did agree on a great many points. We agreed that the results of the work that has been reported here tonight, correlated with the researches of others in the same field, have very definitely eliminated certain points of uncertainty and have established certain facts which are very valuable. So, those who have taken part in this conference this afternoon have had a great stimulus and a great incentive to go back home and go to work again with renewed energy. It is a great encouragement to know that there is a group such as this that is making a concerted effort to study the underlying features of dental caries, which must be determined before we can fully understand the disease or go farther in controlling it. I prepared a discussion before I came which may or may not be pertinent, but contains some of the impressions received from the papers, at least from certain parts of them.

The report of the researches by the Pittsburgh group is a most interesting and valuable document. In an analysis of the report it is apparent that these investigators have attacked the problem of dental caries from a distinctly "localist" point of view. Their researches were preceded by a preliminary survey of the literature pertaining to this subject, in which they attempted to distinguish between established fact and unproven theory. They then selected two principal factors in the problem; first, the action of acids in various concentrations on tooth enamel and the effect of buffers in the saliva as a modifier of this action; second, the classification of the bacterial flora associated with dental caries. The chief significance of the reports arises from the fact that the data in both the chemical and bacteriologic

fields coincides with and amplifies the work of other groups who are studying this problem, and helps to establish certain basic facts which are very important to the understanding of the disease. As a contribution of the Michigan group to this discussion, I shall limit my remarks to a consideration of the chemical and biologic phases, while the bacteriologic aspects will be discussed by Dr. Philip Jay.

The Pittsburgh group have shown very clearly a fact which has been long surmised—that were it not for the buffers of the saliva, the teeth would be seriously damaged and disintegrated by the fluids which come in contact with them. Unquestionably the chemical buffer and the organic substances of the saliva protect the tooth from the action of weak acids. It is not quite clear, however, just what part these salivary buffers play in the process of dental caries for if the saliva, with its amphoteric qualities, were constantly in immediate contact with every surface of every tooth, it is reasonable to expect that the acids of fermentation associated with dental caries would be immediately neutralized before they had an opportunity to attack the enamel surface. There are many indications that saliva does not have ready access to those areas on the tooth surface in which active caries is taking place. Various types of films, bacterial overgrowths, and food debris interfere with ready movement of salivary fluid into that area. We have observed, in the use of antiseptics in the mouth for the destruction of B. acidophilus, that many potent antiseptics have no appreciable effect when used as mouth washes; but when the tooth surfaces have been cleansed of films and extraneous substances, then the germicides when used as before often produce a remarkable reduction or elimination of these organisms. If these liquid germicides are unable to permeate the immediate environment of caries to reach and destroy the fermentive bacteria, is it not reasonable to expect that the salivary fluids are also thus prevented from freely diffusing through these areas? It is for this reason that we raise the question as to the manner in which the saliva and its chemical constitution may control the carious process.

This question is further emphasized by the fact that we have made very intensive chemical studies of the saliva and blood sera of two groups of individuals, one of which was decidedly immune and the other extremely susceptible to dental caries. In them we looked especially for some demonstrable difference in the calcium and phosphorus content, the pH and CO_2 values, and the diastatic action of the saliva. In our results, which as yet are incomplete and unpublished, we have found no distinctive differences in the calcium, phosphorus or pH in either the saliva or blood sera of caries immunes over those of the susceptibles; that is, we do not find that the

salivas of immune individuals carry constantly more calcium or more phosphorus, nor are they more alkaline, than those of caries susceptibles. These data accord with those in the recent work of Karshan of Columbia. Therefore, we are led to the opinion that whatever controlling influence the saliva may have on the caries process is not exercised by its calcium or phosphorus content, or its pH values. It is our opinion that the salivas of both caries-immune and caries-susceptible individuals exert a more or less protective action upon the teeth, and that if the saliva of either type could be kept constantly in immediate contact with every surface of every tooth there would likely be no dental caries. It is only in those areas from which the saliva is excluded more or less completely by films and other factors in the environment that the action of caries begins. Therefore, it seems to us that the next step in this direction is the study of what takes place in the immediate environment of the carious lesions. What is the nature of the bacterial plaque? What types of plaques are favorable to dental caries, which are unfavorable? What is the immediate life history of the caries process in contact with the tooth? These questions deserve consideration; their study offers very serious difficulties.

The authors speak of a phenomenon previously observed: when areas on the enamel have been attacked by caries, and caries has subsequently been arrested, these areas are apparently less soluble in acid than normal enamel. It has been inferred by some that the increased resistance of these carious areas expresses a reaction of the tooth against caries, thus arresting its progress. Such a conclusion is not altogether justified. In the first place, in all such areas in which initial caries is arrested, there has been a change in the environment—either an extraction of an adjacent tooth, the opening of an approximal space, or some other factor—which has facilitated movements of food masses across the carious area, thus cleansing it and breaking up the localized caries process. Subsequently the white area of approximal caries may change to a dark brown or even black, indicative of the precipitation into this partially decalcified enamel of either extraneous substances, or possibly of material from the interior of the tooth. Enamel which has undergone this secondary change, subsequent to the arrest of caries, is often apparently somewhat more resistant to caries than normal enamel. A corollary to this action is seen in occlusal caries, in which it is frequently observed that, when the distal or mesial wall of a cavity breaks out and food in mastication traverses that surface, the caries process is arrested in those areas of the cavity that are cleansed in this manner. Subsequently the soft carious dentin becomes hard, almost flint-like in its characteristics—a secondary change after the arrest of caries. In such cases it is often noticed that this

eburnation does not occur in all parts of the cavity for, in its sheltered portions beneath ledges of enamel where the food does not reach, caries continues actively as before. For these and many other reasons we believe that these changes in arrested caries are not primary reactions of the tooth, and are not responsible for the immunization against caries. Rather are they secondary changes subsequent to the disturbance of the aciduric environment that produced the original lesion. This is quite a different interpretation from that stated by various observers, and agrees perfectly with other known facts related to dental caries.

In our study of caries immunes and susceptibles we have been able to demonstrate only one characteristic difference; namely, the presence or absence of the activity of aciduric bacteria in the mouth. This observation has been very impressively substantiated by the report of the Pittsburgh group. In our recent studies, which are unfinished, there are indications of differences in the CO_2-carrying power, and in the diastatic action, in that the CO_2 is higher in immunes and the diastatic activity is lower. These indications may or may not be supported by our final observations in the study of these groups; but if these differences exist, we would then have to determine their significance and the part they play in the process of caries. Other than these we have, as yet, been unable to determine any characteristic biochemical differences between the blood and saliva of these two types of individuals.

In view of the fact that the saliva constitutes the chief environmental medium which bathes the teeth, it seems strongly probable that whatever controlling influences exist in the mouth to determine the activity of the aciduric bacteria are resident in and carried by the salivary fluids. What these principles are, their nature or their modes of activity, we do not know. As far as we can see, they are not due to the calcium, phosphorus, or pH values. Much work must be done to determine what salivary constituents determine that bacteria may grow luxuriantly in one mouth to produce extravagant caries, and inhibit or impede their action in another. In our further consideration of dental caries, we must also give more attention to the nature of the bacterial plaques in contact with the tooth, and to the part played by the keratinous enamel cuticle, which has been demonstrated by German observers to be insoluble in both acids and alkalis. In regard to this enamel cuticle: possibly when it is present the initial step of caries is not due to acid producing bacteria, but to some form of ferment that is able to digest this protective covering and pave the way for the action of acids. These are some of the many important factors in this intricate problem requiring study before we shall have an adequate concept of the etiologic factors in this very complex disease.

Dental caries is a most unique disease, the like of which does not occur in any other part of the body. At present there is general concurrence in the view that dental caries is an infective process in which specific types of bacteria, acidogenic and aciduric in nature, are resident on the surface of the affected tooth and by their acid production destroy the tooth. These localized infective processes are not in immediate contact with the blood serum or the natural protective forces of the body. They constitute non-pathogenic parasites in the oral cavity but, in the early stages of caries at least, they do not have a parasitic action on the tooth itself. They live, rather, as a reaction to their environment (i.e., the saliva and food debris). They have no direct pathogenic action on the tooth, but produce their specific effects by means of acid products of fermentation. We can conceive, therefore, of this process as being the attack of an extraneous infective agency outside of the tooth and outside of immediate contact with the body protective fluids, and controlled only through the oral environment—saliva and retained food materials. If this concept is true, the control of dental caries must lie in two general fields: the saliva and its action upon dental caries, and the quality of foods ingested and the means of diminishing their retention in the oral cavity.

In the study of saliva we are taken far afield into all those general constitutional conditions that determine its nature and characteristic action. These include heredity, general health, nutrition, immunologic properties, and perhaps other qualities we do not anticipate. The second phase involves the physical and chemical quality of the foods ingested, especially so far as retention and carbohydrate content are concerned, and all those prophylactic measures that determine the amount of food material that constantly remains in contact with the tooth tissues. It is our opinion that through chemical, immunologic, and dietary studies, directed toward a fuller understanding of the salivary principles which control the activity of caries, the greatest benefit will come. At the same time, we should not neglect a consideration of more effective local measures—prophylaxis and dietetics—by which the mouth may be kept free from the essential factors of caries.

It is by such studies as these reported by the Pittsburgh group, correlated with the work of other groups, that progress may be made. The authors of this report are to be congratulated on the vision they have had in the analysis of the problem, and on the thoroughgoing way in which they have attempted to establish certain facts in this study. The combined efforts of those who are especially interested in this problem, in conferences such as is being held at this time, will be of immeasurable benefit in extending the frontiers of our knowledge of this very important disease.

E. H. Hatton, M.D. (Chicago): If I assume that my part in this discussion is to simplify it by picking out only three items for emphasis, probably I shall be able to make the best use of the time allotted to me. From Dr. Friesell's masterly summary of the clinical behavior of dental caries I have selected one fact that I think is worthy of repetition and emphasis. Typical caries occurs in natural teeth inserted into artificial dentures, indeed even in teeth carved from ivory and bone. Not only that, but also in the same locations and under the same conditions as caries in vital teeth. This and similar facts laid the foundation for the development of a method for the control of dental decay that has stood the test of years. Wherever the dentist can convert susceptible areas on teeth—they are well known—into nonsusceptible regions by the use of fillings of gold or other inert substances, and has carried the fillings into the parts of the teeth that are rarely attacked by caries, as far as that tooth is concerned, decay is cured and controlled. May I compare this problem of the treatment and control of dental caries to that of transportation? In the beginning man carried his burdens and himself on his own legs. Next perhaps came the wheel and the sled, and then he trained animals to pull his loads. Boats, sails, steam engines and railroads arrived in due time, and now we have the automobile and the airplane. But none of these later forms of transportation have replaced entirely any of the earlier methods. And so it will be with dental decay, the method evolved by the dentist will always have a place. Wherever we go from this point in the control of dental caries, this fact cannot be overlooked. It is my contention that until now the dentist and the dental investigator have done by far the best job in the treatment, cure, and control of dental caries.

It is my impression that caries is entirely a matter of the environment of the tooth. As Dr. Bunting has clearly stated, two factors are concerned; namely, the composition of the saliva and what enters the mouth, as food and bacteria. I am willing to admit that vitamins, such as C and D, produce tremendous effects upon developing teeth, but when the tooth has erupted and is fully formed, except for the pulp, it is beyond influence by vitamins. For one to say that deficiency of vitamin C causes caries because of the effect that such a deficiency has on the tooth, particularly the enamel, seems to be extremely silly. An advocate of the orange-juice method of preventing dental decay, when asked directly: "Is it the vitamin C in the orange juice that is effective,?" answered: "I do not know." Orange juice may be effective in preventing decay whether it does or does not contain vitamin C. Why shouldn't some one experiment with orange juice that has been deprived of all vitamin content? As far as I know, no one has.

It would seem most advisable to do so. I have been interested in a series of experiments with thin films of mucus placed on glass slides. Gastric mucin has been used because it is readily secured. Some reagents remove the mucin smear with great ease, others seem to make it more adhesive. Acetic and citric acids in dilute solutions wash off the mucin very quickly, as do most of the organic acids. However, lactic acid is a noteworthy exception; it has much less ability to remove these mucin smears than any other well known organic acid. This would seem to be a most interesting coincidence from the standpoint of the current theory of dental decay. Many of the substances that are thought to contain vitamin C, such as, orange juice, lemon juice, tomato juice, sauerkraut juice, cabbage juice, apple juice, etc., are quite as active in removing the mucin smears as the pure organic acids. By implication we may assume that anything which removes mucin from glass slides may be of assistance in cleaning the surfaces of teeth that are susceptible to decay. According to this notion the character of the food, or the order or the manner in which it is eaten, has a substantial influence upon the occurrence of dental decay without any reference to its vitamin content, owing solely to a change in environment of the tooth.

In a recent review of the subject of dental caries in *Die Fortschritte der Zahnheilkunde*, Türkheim compares some figures on the frequency of caries (percentage of carious teeth) in pregnant women with the data for a control group. Group I, age 20 to 25 years: pregnant women, 19.7 percent; non-pregnant, 19.9 percent. Group II, age 26 to 30 years: pregnant women, 27.2 percent; non-pregnant, 26.9. Group III, 31 to 36 years: pregnant women, 32.7 percent; non-pregnant, 32.3 percent. The average is just slightly in favor of the pregnant woman. The only conclusion that the authors could draw from these data, which they had laboriously collected, was that the total number of carious teeth increases with age.

In closing may I remind you once more that until now the dentist has done the best job in the field of the prevention and treatment of dental caries, and that the method he has evolved will always have a place in caries prevention. We must never lose sight of the clinical picture of dental decay, no matter how enthusiastic we may become about some new measure of prevention. I will agree heartily that diet is "in the picture," but only in so far as it affects environment. But any theory that does not explain all the clinical facts that are known about caries is invalid and unacceptable.

I should like to join Dr. Gies and Dr. Bunting in congratulating the authors of this paper, and to thank very heartily the Pittsburgh group for this opportunity to meet with them.

F. E. Rodriguez, B.Sc., D.D.S., Dental Corps, U. S. Army (Camp Holabird, Md.): In dealing with a scientific contribution of a magnitude such as this, it is practically impossible to discuss in detail, and therefore justly, the various findings. I wish to express to the essayists my deep admiration for the unprejudiced and logical method of approach to this most difficult and greatest of all problems of dental pathology—and for the immense labor required for the completion of so broad a research. In recent years, I have not seen a review and analysis that was as comprehensive as that in the first part of the paper. Nor have I seen a more capable scientific discussion and evaluation of the various current hypotheses and findings in this field. Were it possible for dental investigators in general to adhere rigidly to this declaration of fundamental clinical facts and scientific principles, so soundly expressed in this paper, we might gradually put an end to the uncertainty now prevailing, and thus more speedily attain final solution of this problem.

I wish to state my pleasure at the acquisition, by the particular scientific creed with which I am affiliated, of this group of distinguished scientific personnel. Various schools of thought, whose only claim to public attention is based on gross violations of well established tenets of histological and clinical evidence, crowd the dental stage today to such an extent that even scientific ostracism has threatened the supporters of the infectivity view of dental decay. One of the reasons for this attitude has been the marked indifference of modern dentistry to further developments and amplifications of the original Millerian theory as it relates to the mechanism of enamel dissolution in fully formed teeth. For thirty years after Miller propounded his views there was more or less indifferent acceptance of his views and little action. This was true despite the fact that two of the great luminaries of that day, Black and Leon Williams, in accepting and submitting further proof of the principle involved, invited attention to the existing deficiencies, and stated the necessity for further serious research—as Miller himself had done, they showed the avenues that subsequent research should follow. The soundness of the foundation so laboriously built by these three great pioneers of scientific dentistry has stood the test of clinical experience. In fact, modern operative technical procedures are possible only through a recognition of the principles laid down by these men. Such principles are based on the bacterio-chemical nature of dental caries.

When, a little over ten years ago, the bacteriology of tooth decay received renewed attention in the light of modern chemical and bacteriologic laboratory methods, a large block of the dental scientific world was enthusiastically engaged in acclaiming the advent of new concepts of nutritional chemistry in its possible relation to dental disease, and somewhat ruthlessly discarding

the previous fundamental views of the mechanism of enamel pathology. In but few other periods in the history of science have factual basic concepts been thrown aside in an effort to influence quickly, and to dominate, the clinical mind. The result has been the befogging of true scientific issues with the most preposterous interpretations and deviations from the accepted tenets of histology and clinical experience, and the creation of a general professional misconception of the true significance of the new doctrine of local infectivity. Consequently, very few individuals in this country or abroad have had the temerity, in the face of general opposition or indifference, to take up seriously the study of the local bacterial causes of tooth decay in its newer aspects. Among the few American investigators there stands out prominently the name of Russell W. Bunting of the University of Michigan, to whose gallant championing of this cause, backed by the most unassailable evidence ever presented in support of the local factors in dental decay, we owe in a large measure the renewed interest in this great question. And this evening a new brilliant chapter has been added, by Drs. Enright and Friesell and the Pittsburgh research group, to our better understanding of this perplexing problem.

I am very favorably impressed by the experiments on artificial decalcification of enamel and the truly remarkable lesions produced. Years ago I published a report bearing on a similar matter, but my results were far from consistent; and no histological examinations of ground sections of the enamel proper were carried out. It was my belief, at that time, that lactic acid solutions, similar in pH values to those obtained with broth cultures of dental strains of the lactobacillus of caries, could not produce rapid, deep lesions; that is, lesions involving both the enamel and dentin. I was then looking, of course, for gross pathological effects. The disclosures by the authors, following examination of slight etchings of the enamel of immersed teeth, after their careful technique with silver nitrate staining, is a most remarkable contribution to our knowledge of artificial enamel decalcification. I feel somewhat disappointed by the failure of the authors to carry out similar decalcification tests, for purposes of comparison, with cultures of acidophilus from oral sources. The inability of the authors to produce any detectable lesions in certain types of teeth, even after long periods of immersion in organic acids, interests me greatly since such has also been my observation after submersion in broth cultures of L. acidophilus. I have in mind a tooth that withstood for over ninety days the acid onslaught of a broth culture of the lactobacillus maintained at a pH 2.8. The cause of this resistance may find a partial explanation in the presence of the insoluble organic coatings which the authors of this paper report. It is also probable that the varia-

tions in keratin content of tooth enamel, as recently reported by Rosebury and also by Bibby, may have to be considered in this connection. The demonstration by the Pittsburgh group of this ability to resist decalcification is an outstanding observation worthy of the most careful attention in future research.

The application of the solubility-product-constant concept ("K s.p.") to salivary chemistry is exceedingly interesting. As the authors themselves have pointed out, the most significant implications of this concept involve areas the conditions of which are as yet undetermined, inasmuch as the product of the $(Ca^{++})^3 \times (PO_4^{\equiv})^2$, under or adjacent to any particular carious area has not yet been evaluated. This point is of great significance, for the chemical equilibria under or adjacent to a carious area, or bacterial plaque, may be quite different from that in saliva at an area removed from a carious one. Therefore, a demonstration of what happens chemically in saliva gives no direct information as to what may happen chemically at a carious area.

The authors have given the bacteriological phases of this study carefully detailed attention. The endeavor to establish a classification of types based on fermentation, agglutination, and dissociation characteristics has been most thorough and represents, in my judgment, the most authoritative effort to date. The final and acceptable taxonomic groupings or sub-groupings remain as yet in the laps of the gods. It is quite probable that further studies of bacterial dissociation will present the key to the type representing true specificity. For years it has been my opinion, based on personal study and observation of many thousands of strains of both dental and intestinal origin, that we are dealing with a very complex group—a condition comparable to the status of the gram-negative bacilli in the intestines of man and certain animals before the separation of the typhoid, paratyphosis, and coli types, but, as in the latter instance, capable of eventual taxonomic classification. In my early efforts I attempted a classification along the arbitrary lines of morphology as a working hypothesis, but I have yielded to the consensus of opinion that no differentiation can as yet be safely made from the intestinal types. In this respect, the finding of the authors, that Y-type acidophilus strains behave like Group-I dental strains, is an important contribution to the field of pure bacteriology—and, if you will allow me a little presumption, a partial vindication of my particular school of thought.

The report that of 85 patients exhibiting caries 61 percent had lactobacilli, and that of 63 patients without caries 52 percent showed also the presence of the bacilli, seems a little inconsistent in the light of my personal experience. Differences in technique may account for the disagreement, but my per-

centages of bacteriologically positive in active cases of the disease, and, vice versa, bacteriologically negative in non-carious or treated cases, have always been much higher than those reported by the authors. In this connection I notice that the authors put down as positives all acid-broth cultures showing gram-positive bacilli, but failed to state in the paper what confirmatory tests were used to prove the actual presence of the specific bacilli. I find myself in further disagreement with the reported findings of positive and negative mouths when I consider the notable observation by the authors that the Y-type of colony seems to prevail in dental caries. My own studies of dissociation phenomena relating to L. acidophilus indicate that the dissociated, typical-colony forms which are generally rough in topography, but capable of various classifications from slightly granular to fuzzy and then to extremely degenerated and filamentous surfaces, do not possess the pathological significance of the smooth type. These facts must be considered in acidophilus examinations of the dental zone. We do not know what salivary biochemistry brings about this loss of characteristics and qualities, but accumulating evidence points to a lack of acidogenic properties and of general aggressiveness in such forms.

The lack of a practicable method of quantitative examination compelled the authors to make use of the standard qualitative procedure of examination indicated in *table 6*. Qualitative methods in such examinations are fallacious in that a case having only a few inconstant bacilli is placed in the same category as one having a massive acidophilus flora. My quantitative examinations have confirmed my original theoretical view that massiveness of oral invasion—that is, maintained overgrowth— is the determining factor in enamel deterioration. It is my judgment that, irrespective of the well known concepts in the field of the general infectious diseases, the oral fluid in states of general good health, aided by a fair degree of mouth hygiene, is capable of protecting enamel successfully against slight acidophilus invasions. That the authors did not trust, or consider as sufficiently weighty evidence, their original examinations by qualitative methods is shown in their most acceptable and well planned "follow-up" examinations from a clinical standpoint. This step was another indication of the thorough and truly scientific endeavor characterizing the paper in all its aspects. The so-called "follow-up" method, as conceived and carried out in this investigation, is a procedure that surely fills a gap created by the deficiencies of the techniques in general use. In my judgment, the results and analyses of the "follow-up" study of fifty cases is one of the most masterful arguments in support of the etiological relationship of these bacteria to dental caries.

The evidence presented by the essayists to support the views of the

etiological relationship of L. acidophilus to dental caries should remove the doubts in the minds of a large group of the profession. Not only do we find our house of scientific bacteriological endeavor put in good order by this study, but also our general fund of knowledge has been enriched in a most remarkable manner.

Philip Jay, M.S., D.D.S. (Ann Arbor, Michigan): It is gratifying to see so much attention given, in this important report by the Pittsburgh group, to the rôle of Bacillus acidophilus in the etiology of dental caries. Rodriguez, McIntosh, and the Michigan group, at a time when the world was swiftly becoming vitamin conscious, emphasized the significance of the lactobacilli in dental caries. These important contributions were received by dental minds much like water on the proverbial duck's back. Although it is true that very impressive strides have been made by the application of new developments in nutrition to medical problems, we are now beginning to realize that these new principles are not necessarily applicable to dental caries in the same way, since the tissues involved are entirely different. It is to be hoped that this most recent contribution to the accumulated evidence of the importance of B. acidophilus in dental caries will serve to stimulate enthusiastic research for methods of controlling overgrowths of this organism in the mouth. Such investigations would probably proceed along three lines: drug therapy, manipulation of diet, and immunology. The authors intimate that they are now attempting to control the growth of the lactobacilli in the mouth by the use of bactericidal substances. My own attempts to this end have been disappointing, although our studies along this line have not yet been exhaustive. At one time it seemed likely that hexylresorcinol and metaphen might be useful in the control of dental caries. In isolated cases these drugs appeared to be very effective. But, in a controlled experiment reported by us in 1930, we found that our control group fared better than the individuals who received prophylactic treatment with the drugs—69 percent of the treatment-group had new caries during a 10-month period, whereas only 50 percent of the controls, with no treatment whatever, developed new caries. It might be added that the portion of the treatment-group on metaphen received an average of forty-eight thorough prophylactic treatments during the period of observation. However, there still remains the possibility that an effective therapeutic agent will some day be discovered.

There are indications that caries may be controlled by the adoption of certain diets. Although the evidence presented has not been conclusive, there is agreement among many investigators that favorable results have

been obtained on various diets. The phenomenon concerned is difficult to explain because of the peculiar structure of the enamel, and the constancy in the amounts of calcium and phosphorus in the blood and saliva of both susceptible and immune individuals. There remains, however, the possibility that the bacterial flora of the mouth may be very definitely altered by variations of the diet. This was clearly demonstrated to us in a group of institutionalized children where B. acidophilus and dental caries were practically eliminated after the adoption of a diet low in sugar but otherwise thought to be well-fortified. Despite the fact that caries was effectively controlled in this institution, a more recent nutritional analysis indicates that this successful diet was deficient in many respects. We know with certainty only that the children in this institution are remarkably free from B. acidophilus and that dental caries is negligible. However, in the event that a potent dietary principle should be discovered, its application to the average individual would still remain a serious difficulty.

If there is general agreement that bacteria are the immediate cause of dental caries, there remains the immunologic approach. Here again the entire principle of immunity is atypical. The active immunologic factors to be effective must, in this case, be antibacterial rather than antitoxic. These forces must reach the microörganisms indirectly rather than directly through the blood stream, since dental enamel is not a vascular structure. Nevertheless, there are indications that this phase of the problem should be thoroughly investigated. We have found that caries-susceptible individuals react locally to intradermal injections of minute amounts of B. acidophilus filtrate, while caries-free subjects, as a rule, do not. These reactions cannot be correlated 100 percent with dental caries, but it is not altogether improbable that the exceptions fall within the limit of error in the diagnosis of active caries at the time of the skin test, a difficulty with which the Pittsburgh group is probably well impressed. Examination of the blood serum from susceptible and immune groups revealed that sera of immune individuals contain B. acidophilus agglutinins in dilutions as high as 1/640, whereas sera from susceptible subjects either failed to agglutinate B. acidophilus antigen at all or only in extremely low dilutions. We have also seen indications that the titre of the susceptible serum can be raised to the level of the immunes by the use of a polyvalent B. acidophilus vaccine. This was accomplished in two out of five attempts. In both cases the skin reaction became negative, coincident with the appearance of B. acidophilus agglutinins in the blood stream. These studies are being elaborated and more information will be forthcoming.

To return to the report under discussion: we find very little that is not

in agreement with the bacteriologic work done in our laboratories. It is evident that the Pittsburgh investigators have been impressed by the pleomorphism of the organism under investigation. They too have noticed the points in similarity between the mouth and intestinal types. Although comparison of fermentation classifications is sometimes difficult, it is very apparent that Groups I and II of the Pittsburgh classification coincide with the Michigan Groups 1 and 2. The similarity of the fermentation reactions between Y-type intestinal-strains and the Group-I dental-strains accords with the findings in our laboratory. The failure of the Pittsburgh workers to dissociate their strain 114 into the rough type is difficult to understand, unless it was because the transfer from the rough marginal outgrowth of the smooth colony was not again made directly to agar. It is likely that the return to broth at this point was responsible for the reversion to the smooth type. In our laboratory, these rough outgrowths are picked with the greatest care under a dissecting microscope to eliminate the possibility of including any of the smooth portion of the colony in the transfer. It is much more difficult to obtain typical smooth colonies from the rough forms, although we have succeeded in obtaining fairly smooth colonies from the Rettger strain 43. The Scavino strain was more stable.

It is particularly important that the Pittsburgh investigators were also able to demonstrate the presence of B. acidophilus prior to the clinical manifestation of dental caries, the caries appearing subsequent to the invasion of B. acidophilus. This type of evidence precludes the possibility that B. acidophilus is a secondary invader, as has been repeatedly stated by Hanke.

Another point of difference between our studies is that we frequently find yeast and Streptococcus fecalis in our examinations of both immunes and susceptibles. I did not see them listed in *table 6* with the other organisms isolated from the mouth. The authors also state that by the use of a pH 5.0 broth, the streptococci and oidium-like organisms are eliminated. We have not found this to be altogether true for, in our cultures taken in 1 percent dextrose meat-infusion broth pH 5.0, staphylococci, streptococci and yeasts have occurred with great regularity. It is true, however, that after long periods of incubation, the aciduric rods were often the sole survivors. The fact that this medium was not too differential gave us the opportunity to notice that the streptococcus appeared in all mouths regardless of the occurrence of dental caries, whereas B. acidophilus was conspicuously absent from caries-free mouths.

I have tried to find points in which we disagree, but you may observe that these have been few. I cannot close, however, without calling attention to one point that for a long time has been a source of annoyance to many of us.

I refer to the time worn thought that bacteria are not responsible for the onset of dental caries because many dirty mouths do not have dental caries whereas, on the other hand, teeth that are apparently clean do decay. This observation is quoted in the Pittsburgh report among the few objections to the Millerian theory of dental caries. There is nothing at all remarkable about this fact. All bacterial types are not synonymous with filth. All unclean people do not have tuberculosis or measles, nor are all victims of tuberculosis and measles dirty. We are concerned in dental caries with a specific organism. It may or may not occur in an unhygienic environment, and it may or may not occur in the cleanest of mouths. On numerous occasions we have examined mouths that were free from dental caries, and also most disagreeably dirty, but in which we repeatedly failed to find B. acidophilus. Mouths with active pyorrheal lesions are known to be teeming with bacteria, but, as a general rule, examinations for B. acidophilus are negative; and it is a common observation that active pyorrhea and progressive caries rarely occur at the same time. Discovery of the factors that determine a favorable environment for B. acidophilus is our goal. It need have nothing whatever to do with personal filth.

Theodor Rosebury, D.D.S. (New York City): I shall discuss this paper from the only viewpoint open to me—that of an investigator actively engaged in the study of dental caries, along lines which happen to be very similar to those followed by the authors. I cannot forbear, in passing, to express my appreciation of the authors' statements relating to the clinical aspects of dental caries. These statements constituted a clear and forceful exposition of a subject needing just such treatment. There has been an apparent tendency in the recent literature to overlook clinical facts, and to treat the subject of dental caries academically, or in complete disregard of clinical experience, which may well be said, even now, to comprise the most fundamental and most clearly established phase of the subject.

The manner in which the authors dispose of the subject of diet in relation to caries is somewhat misleading. The authors present the following conclusions from the literature: (a) diets rich in vitamin A, C, and D and an alkaline ash, utilized during the developmental period of teeth, produce teeth less liable to decay; (b) a continuation of a similar diet during the post-eruptive stage of teeth produces saliva with some protective power against caries; and (c) deficient diet is important only as a predisposing factor in dental caries. Not one of these conclusions is actually substantiated by the literature. Each is little more than speculative; the evidence that now appears to support these contentions may eventually fit neatly into some fundamentally dif-

ferent concept. We can say little beyond the statement that diet plays a part in dental caries; how large, how small, cannot be said with any certainty; nor can we say whether the determining factor in diet is a deficiency, or an excess, of some one or several ingredients, or a matter of physical rather than chemical character.

The reasons advanced by the authors for not attempting an experimental study of caries in animals are also not entirely clear. The well known caution required in the interpretation of clinical findings on human subjects can hardly be too often repeated: so many experimental factors, readily controllable in animal work, are unavoidably beyond control in clinical studies that interpretations of clinical findings must be made with great care, and nearly always with reservation. Statements such as those of the authors regarding millers, bakers, and Eskimos have been made to prove mutually contradictory points—we are, after all, not justified in correlating, *a priori*, the caries of millers with the carbohydrate content of their diets, or the absence of caries in Eskimo with their meat diet. So many other factors, of which we know nothing, must be present in both! Working with animals, these objections need not enter—we can provide conditions; and if we cannot control them completely, we can at least observe the uncontrolled with the controlled. The objection that dental caries as noted in animal experiments has not been sufficiently similar to the lesion in man, to warrant conclusions about human conditions, has been a valid one, and certainly applies, as the authors state, to Howe's work with guinea pigs. Very recent observations seem, however, to have invalidated this objection. We have lately produced in rats, in confirmation of earlier work by Shibata and others, dental caries so closely simulating enamel-fissure caries in young human subjects that these animals can now be used for controlled experiments to increase knowledge of the disease in man. From our own viewpoint, particularly in the light of these recent findings, this phase of dental-caries research is perhaps more important than any other.

One other point in the authors' review of the literature relates to the statement of Hanke's that the spirochetes associated with pyorrhea are acid-producers. A paper by Akatsu, published in 1917—so far as I know the only paper in the literature on the subject of carbohydrate fermentation by spirochetes—indicates that *Treponema microdentium* may produce moderate amounts of acid from glucose and glycogen; but there seems to be no other evidence to warrant Hanke's statement. Of the spirochetes associated with pyorrhea, apparently the preponderant types are Vincent's spirochete, about the fermentation reactions of which nothing seems to be known, and Noguchi's *Treponema mucosum*, which Akatsu studied, but about whose

acid production he says nothing. In general, the statement that spirochetes are acid producers is apparently not warranted by present knowledge.

The authors state that in teeth with incipient enamel caries, the carious enamel, being covered with exposed enamel keratin, or organic accretions, or both, is more resistant to the action of acids than is the unaffected enamel. Bibby, in a recent publication, describes a similar phenomenon, observed in celloidin-decalcified sections as "consolidation" of the enamel keratin, which he considers to be protective. But a question arises. Bibby states that his specimens were fixed in Zenker's fluid. The authors, in another connection, speak of the use of formalin for the preservation of the teeth used in their chemical tests. While I know of no evidence that would specifically contraindicate the use of fixing agents in this connection, their use should be avoided on principle, or otherwise the possibility that artifacts might arise from such use should not be overlooked. In the instance in question, fixation before subjection of the teeth to the acid might have brought about an artifactual toughening of the enamel keratin exposed by caries, though failing to penetrate normal enamel; and might have not only rendered this physically delicate tissue less easily broken, but also produced the "consolidation" described by Bibby, and thus limited the penetration of acid subsequently applied. The authors do not indicate that this possibility was tested. I have completed a simple test of this matter, and am glad to be able to confirm the authors' findings. The six teeth I now present were freshly extracted, and kept in physiological saline for periods not exceeding seven hours before being used. Two were then placed in fresh saline, two in 4 percent formalin, and two in Zenker's fluid; left forty-eight hours; washed in running water for an hour and a half; and placed in *N* lactic acid for twenty-four hours. All these teeth show the phenomenon described. Microscopic examination of the chalky material, left relatively unaffected by the acid, indicates that it is partly decalcified enamel. [No photographs presented for publication.]

The classification of lactobacilli offered by the authors, based on fermentation and agglutination reactions and growth at a low temperature, is the best, involving the oral strains, that has yet appeared. Attempts to classify these organisms in the past have resulted in groupings that were artificial rather than practical, and which had no actual taxonomic significance. Our own attempts to classify them on morphological grounds, following the leads of McIntosh *et al.*, Rodriguez, and Bunting and his co-workers, have not yielded satisfactory results. Our tentative classification on the basis of acidogenic powers, while of immediate usefulness in the investigation itself, lacks both the rigidity and the correlation with other characteristics that might make it useful in a wider sense.

On one point relating to this matter, however, a word of criticism is in order. It appears that all the strains of lactobacilli examined by the authors had been growing artificially for a year or more—that is to say, were "old" strains. The greater part of our work has been done with relatively fresh material—strains growing artificially for periods up to six months. Our studies indicate that an "old" group is notably more homogeneous than a "young" one, in part apparently because in "old" growths original types tend to coalesce, and partly because some of the more aberrant types tend to die out. This circumstance may account for the relatively slight variability of the authors' strains, as compared with the extreme differences encountered in a fresh batch, where frequently each strain appears as a type unto itself. This circumstance does not invalidate the authors' classification, which may be said to hold at least for stable strains; but it does seriously interfere with attempts to correlate strain characteristics with incidence or activity in dental caries. If all strains of lactobacilli are biologically related, we might reasonably expect that all will converge toward a common type when cultivated for a sufficiently long period under uniform conditions, since we know of no other cause for their initial variation than differences in their original environment. But we should not assume that strains artificially altered in this way represent the organisms at the original source.

With regard to the matter of dissociation: we have studied the surface-colony forms that appear in routine cultures, without however attempting to force dissociation. Our results show that colony-form variation is very great; also that the colony-form of individual strains is by no means stable, but undergoes considerable change, particularly in freshly isolated strains. I submit for your inspection a series of photographs, all of 48-hour streak cultures on 1 percent glucose agar. [Not presented for publication.]

The most important phase of the work described tonight consists, I think, of the authors' attempted correlation of the biochemical properties of lactobacilli with factors influencing solubility of enamel in acids. Their conclusion that a pH of 5.0 represents a limiting value in both relations—the limit below which bacteria other than lactobacilli cannot survive, and above which, under conditions assumed to approach those which operate *in vivo*, enamel does not dissolve—is so striking that it merits most careful examination. At Columbia we have also been interested in finding the relation, if any exists, between enamel solubility and the acidogenic powers of lactobacilli, both in terms of pH. Thus far we have not been able to satisfy ourselves either that lactobacilli alone among bacteria are capable of decalcifying enamel *in vivo*, or that any critical pH level of enamel solubility exists. The problem presents aspects of great complexity, and seems to demand the

utmost in quantitative technique. The possible agency in caries of bacteria other than lactobacilli cannot be altogether excluded. The authors' statement that "the only microörganism commonly found in the food debris, in direct contact with progressive caries of the enamel, that can tolerate and produce additional acid in an environment below pH 5.0, is a lactobacillus," agrees with the well-established circumstance, with which our own findings are in complete accord, that lactobacilli are the only organisms from carious teeth which grow consistently in broth at pH 5.0. This finding must have significance in this connection; but it should not be taken as evidence that other mouth organisms cannot survive this level of H-ion activity, for our results and those of others indicate the reverse. We find that lactobacilli, under optimal conditions, are able to reduce the pH of the medium to, and to survive at, a level approaching pH 3.0. Other common fermenting bacteria (B. coli and Staph. aureus) under similar conditions produce and withstand acidity approaching pH 4.0. The results of other workers (see the table in Appleton and Bryant: Laboratory Manual of Bacteriology, pp. 35–36) indicate that a number of organisms, among them some which occur in the mouth, are able to withstand levels of acidity well below pH 5.0.

Our own results, and also general considerations, make me somewhat skeptical of the authors' interpretation of their findings in the matter of acid solubility of enamel. The authors state that in the experiments to determine the critical level of pH 5.0, they saturated their buffers, or nearly saturated them, with tricalcium phosphate, and then re-titrated to their original pH levels. It is obvious, of course, that these buffers were then no longer saturated. I realize that the authors were attempting to reproduce the conditions suggested by Forbes, and that the added acid was intended to represent the product of bacterial metabolism in a medium previously saturated with Ca^{++} and $PO_4^{\equiv}$ ions. But, reasoning on the basis of the titration curve of lactic acid with sodium hydroxide, in which pK = pH = 3.85, and an abrupt rise toward the alkaline occurs at about pH 4.7, the amount of lactic acid finally added to restore the level of the lactate buffer to pH 5.0 was probably disproportionately small, and may have been rapidly exhausted, with the production of very little effect on the enamel. Furthermore, the authors' results indicate that, in the systems containing Ca^{++} and $PO_4^{\equiv}$ ions, a degree of solution of enamel occurred in the lactate buffer at pH 5.0, and in the citrate buffers at considerably higher levels, even to pH 8.0, in sixty days. When to these considerations is added the circumstance that we may not be justified in assuming that the conditions employed by the authors approximate those *in vivo*, the value pH 5.0 does not seem to have the critical significance the authors assign to it. Consequently, al-

though I regret the circumstance, the important question of the possible interrelation of specific bacterial activity and enamel solubility still seems to be open.

In conclusion I must express my cordial appreciation of the splendid spirit the authors have shown in inviting and welcoming this free and open discussion. This meeting represents, I think, one of the finest types of stimulation dental research can receive.

R. E. Morgan, D.D.S. (Pittsburgh): Something has occurred to me which, I believe, emphasizes the importance of Dr. Bunting's statement about the influence of saliva upon dental caries. I read an article in the *Dental Cosmos* by Dr. Prinz concerning seventeen cases in which the saliva was markedly scant. In these patients—I happen to have a similar one under observation now—there is a rapid destruction of the tooth by dental caries. The patient seems to be quite healthy otherwise. The lack of saliva seems to be the main factor in the incidence and rapidity of caries in these cases.

Dr. Enright: The first question raised by Dr. Gies must be answered in the negative. The saliva cannot contribute protection to tooth enamel, through its neutralizing power or common ion effect, on localized spots it cannot reach. As to Dr. Gies' second question: I know of no practical way in which the saliva or its equivalent could be made to protect the areas usually subject to caries.

We agree with Dr. Rodriguez' statement concerning the desirability of a quantitative method for bacteriological examination of the dental zone. In our clinical study, in progress for the past two years, we are using a quantitative method based on that described by Leonard and Feirer.[12] Although this and other quantitative procedures leave much to be desired in the type of evidence they offer, we believe they should be used for the relative information they contribute. In a study of our former qualitative procedure as compared with the quantitative method more recently used, we learned that the qualitative is somewhat quantitative in effect because the method of collecting stagnant-debris samples from the dental zone is sufficiently crude to give negative lactobacilli findings when only slight infection of the dental zone exists.

As to Dr. Jay's disagreement concerning the types of microörganisms in the pH 5.0 media used by the Michigan group and by us, I do not know how much the fact that we used 1 percent galactose whey-broth instead of 1 per-

[12] Leonard and Feirer: *Dental Cosmos*, 1927, lxix, p. 559.

cent dextrose beef-infusion was responsible for our obtaining a higher percentage of pure lactobacilli cultures. I believe that the week of incubation we allowed was a major factor in this regard.

Dr. Rosebury's statement concerning the reason for the negligible solvent action on enamel above pH 5.0 in the lactate-buffer solutions previously saturated with tricalcium phosphate is pertinent. He calls attention to the small amount of free acid in this range. We agree with such an explanation; that is why the experiment was extended to include the citrate-buffer series. In the latter we have solutions above pH 5.0 containing as much free acid as that available in the pH 4.6 lactate-buffer solution. Negligible etching of enamel in the former and marked in the latter indicates, we believe, that pertinent factors other than the amount of free acid operate to give the protection observed in solutions at pH 5.0 and above. The type of phosphate ions seems to play a significant part in this regard. A solution containing a major proportion of $H_2PO_4^-$ might be expected to do more damage than one containing either $HPO_4^=$ or $PO_4^{\equiv}$.

We agree with Dr. Rosebury that the determination of a relationship between enamel solubility and the acidogenic or aciduric powers of certain bacteria presents problems of great complexity, particularly when an attempt is made to simulate conditions in vivo. Only time will tell how much we have succeeded in our efforts.

Dr. Friesell (in conclusion): I am glad to hear the unanimity of opinion expressed by the discussors as to the need for keeping the clinical picture of dental caries in mind in all attempts to solve this baffling problem. Dr. Bunting evidently misunderstood our interpretation of the rôle of the acid-insoluble areas. We do not infer or believe that the increased resistance of these carious areas is an expression of a vital reaction on the part of the tooth against caries.

I am sure that notwithstanding any views the audience may have as to the merits, or lack of merits, of the paper, you will all agree that we could not have selected a finer group of discussors, nor could we have listened to more constructive discussions. The discussors and essayists have been all afternoon in conference on many things related to the paper that were not inflicted upon the audience tonight. This has been a very encouraging meeting. We greatly appreciate having these men spend so much time with us in studying this important subject.

LOCAL FACTORS IN DENTAL CARIES

EDWARD C. DOBBS, D.D.S.[1]

Department of Biochemistry and Pharmacology, School of Medicine and Dentistry, University of Rochester, Rochester, N. Y.

INTRODUCTION

The varying susceptibility of teeth to the action of dilute acids, *in vitro*, was described in 1890 by Miller (1). He placed extracted teeth in fermenting carbohydrates, and produced dental lesions that could not be differentiated macroscopically or microscopically from true dental caries. McClelland (2) immersed carefully dried and weighed enamel in acid solutions, the pH values of which were known. The enamel was again weighed and compared with its former weight. A loss of substance was found in enamel immersed in acid solutions below a pH of 6.5. The loss in weight was inversely proportional to the pH, and proportional to the duration of exposure in the acid solutions.

Paul (3) described a membrane on enamel surfaces, and suggested its rôle was protective to teeth. Mummery (4) described a tissue membrane on enamel of newly erupted teeth. He accredited its origin to remnants of the enamel-forming organ. There were two distinct layers: a thin, non-cellular layer having impressions of the enamel surface, and a cellular layer, both now known as Nasmyth's membrane. Chase (5) found that membranes removed from adult teeth were not Nasmyth's membranes, but films containing bacteria. He considered these films to be bacterial in origin. Black (6), Williams (7), and Miller (1), believed plaque is necessary for the localization of acids of sufficient concentration to produce dental caries. Plaque (film) includes collections of saliva and food debris that adhere to tooth surfaces, and is more or less permanent. Plaque must be differentiated from temporary food collections, from calculus, and from colorless mucin films.

Dental caries begins on the external surface of enamel. The passive resistance of teeth is primarily in this tissue. As this work was

[1] Rockefeller Fellow in Dentistry.

concerned with local factors in dental caries, it was confined to experiments on enamel. The experiments were designed to show (*a*) the effect on enamel of organic acids that may be present in the oral cavity; (*b*) the rôle of dental plaque in dental caries; and (*c*) a correlation of the data with the incidence and control of dental caries.

EXPERIMENTAL

Effects of acids on dental enamel

Freshly extracted teeth, carious and non-carious, were kept in isotonic salt-solution, in a refrigerator. In the tests, they were covered with weak acids. The rate and degree of decalcification are recorded in *table 1*. Only teeth decalcified sufficiently to be scratched with a sharp pointed instrument were recorded. The total percent of teeth having softened enamel is shown for each day in each acid. The organic acids named in *table 1* are produced by fermentation in the mouth. If the produced acids come in contact with tooth tissue, decalcification takes place. Lactic acid in fermenting carbohydrate mixtures may amount to 0.75 percent (1)—about pH 3.3—sufficient at body temperature to decalcify through the enamel in seven days. Acetic acid may reach 0.875 percent, and, at that concentration, decalcifies through the enamel at body temperature in eight days.

The decalcification was not uniform: some teeth had softened enamel; others were unchanged. Areas on the same teeth varied in time and degree of decalcification, i.e., the worn surfaces were first to show softened enamel. These areas, free from visible plaques, were kept clean by attrition from mastication and by excursion of food; and are not usually susceptible to dental caries. A general acid condition of the mouth would affect these surfaces by causing the enamel to soften and thus to increase the possibility of wear during mastication. The contents of the stomach is often regurgitated into the oral cavity during the early months of pregnancy. The chyme contains 0.1 or 0.2 percent of free HCl (8), which would rapidly decalcify the enamel, and thus facilitate food accumulations and dental caries. A weakening of the enamel would occur, predisposing it to fractures. This may explain, in part, the so-called "soft teeth of pregnancy."

Table 2 shows the varying susceptibility of enamel to 0.75 percent

lactic acid. Teeth from ten patients were treated and observed separately. There was a slight variation in susceptibility of teeth from the same individual, and a greater variation in susceptibility of teeth from different individuals. This variation in rate of enamel

TABLE 1

Rate of enamel decalcification of freshly extracted teeth; in percent

ACID		DAYS									
Name	Per-cent	1	2	3	4	5	6	7	8	9	10
Lactic	0.75	44	69	78	78	79	92	98	98	98	100
Butyric	1.0	8	8	17	17	17	25	43	50	59	82
Acetic	0.87	61	100								
HCl	0.1	0	33	60	75	90	96	100			
Succinic	1.0	5	14	47	52	56	69	74	79	91	100
Malic	1.0	0	0	6	13	20	46	55	100	100	100

TABLE 2

Variation in the susceptibility of enamel of ten patients to 0.75 percent lactic acid; in percent

PATIENT NO.	DAYS									
	1	2	3	4	5	6	7	8	9	10
1	28	28	42	42	85	85	85	100		
2	18	18	18	18	45	54	72	90	90	100
3	33	66	75	75	83	92	100			
4	60	80	100							
5	50	100								
6	53	53	65	65	94	100				
7	100									
8	75	75	100							
9	70	100								
10	0	9	68	77	86	100				

decalcification among individuals must be a factor affecting their susceptibility to dental caries.

Areas on enamel that showed resistance to weak acids, *in vitro* experiments, were found to be covered with plaques of sufficient size and density to be distinguished without the aid of a stain. These plaques varied in color—gray or brown; were firmly attached to

enamel; and covered normal enamel, fillings, whitened enamel, and definite carious lesions. The density of plaques varied from a soft adhesive mass to a dense firm membrane.

Experimental investigation of plaques

Plaques were removed by immersing teeth in 5 percent HCl to which a small amount of alum [$AlK(SO_4)_2$] had been added to prevent protein in the plaques from swelling. A diffusion apparatus was constructed, using a 500-cc. jar and a 25-cc. pipette bent at the end to form a J-tube

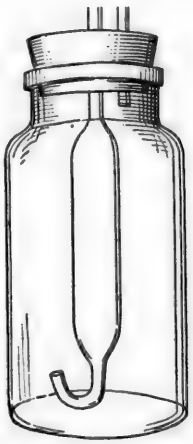

FIG. 1. DIFFUSION APPARATUS USED TO TEST PERMEABILITY OF PLAQUES

(*fig. 1*). This set-up was not what was intended, but the fragility of the plaques required it. The pipette was filled with solution to be tested; outside, in the bottle, was distilled water plus a color indicator for acid-base changes. A plaque was sealed over the end of the J-tube with hot liquid paraffin. Phenolphthalein was used for basic solutions; congo-red, for acid solutions. Passage of acid or basic ions through the plaque membrane was demonstrated by a color change at the point of egress. The results are shown in *table 3*. The control tube contained KOH, 1 percent. A small hole was made in this plaque. After a few hours of no change, movement was continuous until the indicator made a complete color change in about twelve hours. In

the other tubes, with the plaques intact, there was a primary passage of water and indicator into the J-tube, and a slight rise therein in the water level; also a secondary egress of salt molecules. The diffusion continued until the indicator made a complete color change (exceptions noted), when the experiment was discontinued. The area of diffusion surface on the plaques varied from 3 to 7 sq. mm. KOH initiated a color change in 9 to 10 days; NaOH, in 11 to 12 days; lactic acid, in 4 days; Na_2HPO_4 not at all. The Na_2HPO_4 solution was very dilute, only slightly alkaline, and there was not sufficient diffusion to produce a color change.

TABLE 3

Diffusion of salt solutions through dental plaques

SOLUTION		DAYS									
Name	Percent	1	2	3	4	5	6	7	8	9	10
KOH*	1.0	C									
KOH	1.0	+	+	+	+	+	+	+	+	+	C
KOH	1.0	+	+	+	+	+	+	+	+	C	
Lactic acid	0.9	+	+	+	C						
Lactic acid	0.9	+	+	+	C						
NaOH	1.0	+	+	+	+	+	+	+	+	+	+
Na_2HPO_4	0.1	−	−	−	−	−	−	−	−	−	−
Na_2HPO_4	0.1	−	−	−	−	−	−	−	−	−	−

* Control, with hole in membrane. + sign = movement of ions through membrane. − sign = no movement of ions. C = indicator made complete color change.

A quantitative method was used with the same type of apparatus. It was found that 0.01 cc. of 1 percent NaOH or KOH passed through the plaques per day. Lactic acid passed more freely—0.04 cc. of 0.75 percent per day. For the Na_2HPO_4, the Fiske and Subbarow (9) method for phosphorus was used—0.13 mgm. of Na_2HPO_4 diffused through the plaque per day. This amount of Na_2HPO_4 neutralizes only 0.002 cc. of 0.75 percent lactic acid. The slowness of the passage of Na_2HPO_4 through plaques is significant. Salivary buffers consist of carbonates and phosphates. The inability of these electrolytes to pass through plaques, in appreciable amounts, makes the neutralizing value of the salivary salts of little importance in controlling the acids that produce dental caries. Mucin is amphoteric and can neutralize

acids, but its viscosity prohibits its passage in appreciable amounts through plaques. The dense nature of plaques also inhibits fluids in the oral cavity from diluting these acids. Therefore, acids formed beneath plaques are but slightly affected by saliva. The diffusion of acid outward into the mouth is of greater importance.

Dental caries was not found beneath all plaques. Whitened areas of decalcification were found beneath many plaques, suggesting a self-limiting process. This was particularly true beneath thick dense plaques, and might be due to the absence of carbohydrates for acid production, to a change of environment unfavorable for continued growth and development of acid-forming organisms, or to an accumulation of decalcified enamel that inhibits further decalcification. An investigation was made of the first factor by a similar diffusion experiment, with 5 percent glucose solution in the tube, distilled water outside, and a plaque sealed over the end as before. After five days the water was tested quantitatively by the Benedict method for glucose; a total of 16 mg. had diffused through the membrane—3.2 mgs. per day. As the plaque-membrane's diffusion surface was about 3 sq. mm., the amount of glucose passing through per sq. mm. was about 1 mg. per day. Soluble carbohydrates pass through plaques, and under favorable conditions are converted into acids by fermentation.

The last factor above—presence of decalcified enamel tissue at the site of progressive acid destruction *in vitro* experiments—would inhibit the advance of acid destruction. It was found that decalcification progressed, but more slowly than in the initial lesion. Whether this is true in active caries, with bacteria advancing as the tissues are softened, was not determined. It appears that the solubility of the products formed in the process of enamel decalcification is an important factor in the progress of dental caries.

The gross pathology of tooth decalcification presents two pictures. First, an acid decalcification of the tooth tissues, the decalcified salts remaining *in situ*. Second, a dissolution of the decalcified tissue, and production of a cavity. In the first type, the lesion is superficial and apparently self-limiting, although teeth were seen in which the dentin was affected, there had been a pulp reaction, and secondary dentin had been formed, suggesting that this type of decalcification is slow or less destructive to pulp tissue than the more rapid cavity-forming

type. In the second type of decalcification, salts formed by the acids are soluble and diffuse from the site, or are further acted upon by bacteria and changed into soluble substances that diffuse from the site, forming a cavity. This rapid type of tooth destruction is associated with acute dental caries. Both types may occur on the same tooth, the second type always decalcifying to a greater depth than the first.

The affect of an alkaline mouth-wash in controlling acids within plaques was investigated. A similar diffusion apparatus was used: 1 percent $NaHCO_3$ was placed in the J-tube; 0.65 cc. of 1 percent solution of $NaHCO_3$ diffused per day, which would neutralize only 0.065 cc. of 0.75 percent lactic acid. Mouth washes are therefore of doubtful value in neutralizing the acids that produce dental caries.

Solubility of dental plaques

The removal of plaques from teeth by a solvent is of interest from the standpoint of control of dental caries. Plaques were removed from teeth and placed into the testing solutions kept at 37°C. At intervals for three hours the tubes were shaken. The solution effect was slight in all cases (*table 4*). The solubility was determined by making a xanthoproteic test for proteins in the filtered solutions. Any change in density and firmness of the plaque was noted. Neutral salts produced no effect; acetic and basic salts induced a slight solubility of the proteins and a softening of the plaques. Clinically the solvents tried are of no value in removing plaques *in vivo*. Plaques can be easily removed from accessible surfaces with a motor-driven brush and pumice, but only with difficulty from inaccessible surfaces. This explains the inefficiency of the tooth brush in controlling dental caries.

Production of dental plaques in vitro

Attempts to produce artificial plaques on extracted teeth were successful. Teeth, cleaned and polished with a motor-driven brush and pumice, were immersed in saliva and bread crumbs for eight hours at body temperature, when the solutions were poured off, and the teeth allowed to remain at body temperature for sixteen hours. By repeating this process (12 days), teeth were covered with plaques resembling those found in the mouth. No effort was made to control the bacterial

growths. Alternate wetting and drying, plus the bacteria and their products, denatured the mucus from a viscous fluid to a firm gel. The denaturing of mucin was studied by placing small amounts of salivary mucin (precipitated with acid) upon watch glasses, drying over P_2O_5 for twenty-four hours, then removing and quickly weighing. Distilled water was added to the dried mucin for fifteen minutes. A few minutes over this time would not change the weight when weighing to 0.1 mg., so the time lost in weighing was not important. Each time the mucin imbibed less water, and became more dense and firm until a

TABLE 4

Resistance of dental plaques to solvents (solutions kept for 6 hrs. at 40°C.)

SOLUTION		SOLUBILITY	CHANGE IN STATE
Name	Per-cent		
H_2O		Insoluble	No change
$MgSO_4$	1.0	"	" "
NaOH	0.1	Slightly soluble	" "
KOH	0.1	" "	" "
HCl	0.1	" "	" "
Na_2CO_3	1.0	" "	" "
$Ca(OH)_2$	sat.	" "	" "
C_2H_5OH	70	" "	" "
$MgHPO_4$	sat.	Insoluble	" "
Na_2HPO_4	1.0	Slightly soluble	" "
KCNS	0.01	" "	" "
K_2HPO_4	1.0	" "	" "
KH_2PO_4	1.0	" "	" "
$CH_3CHOHCOOH$	0.75	" "	" "

constant adsorption-value was reached in four to six days. This constant was but slightly changed by salt solutions in concentrations present in saliva (*table 5*). Di-sodium hydrogen phosphate, potassium sulphocyanate, and calcium hydrogen phosphate changed the adsorption constant less than 1 percent. Lactic acid increased it 5.88 percent compared with water. The mucin became firm when bathed in disodium hydrogen phosphate, and to a lesser extent in lactic acid, calcium hydrogen phosphate, and potassium sulphocyanate, respectively. The inorganic salts of the saliva have little or no part in the denaturing of mucin.

Dental plaques *in vivo* are probably influenced by the conditions applied in the production of artificial plaques, i.e., during the day, food mixed with saliva collects upon the surfaces of the teeth not kept clean by mastication and prophylaxis. During sleep the mouth becomes dry, bacterial growth is increased, and the collected mucus is denatured, forming resistant plaques.

Contents of dental plaques

Plaques are chiefly organic material. Protein tests—Millon's, xanthoproteic and biuret—were positive. The iodine-starch test was negative. Benedict and Fehling tests before hydrolysis were negative for glucose; after hydrolysis, were positive. This reduction was

TABLE 5

Effect of salt solutions, normally present in saliva, on the imbibition-constant of mucin

SALT SOLUTIONS		CHANGE IN WEIGHT
Name	Percent	
		percent
Na_2HPO_4	0.11	0.93
$CaHPO_4$	0.1	0.69
KCNS	0.01	0.28
$CH_3CHOHCOOH$	0.75*	5.88

* May be present in the mouth.

due, in part, to the well-known cleavage of mucin, with a production of non-fermentable hexosamines (10). The mucin content of plaques is from 27.62 to 52.17 percent. This was determined by dissolving the mucin from plaque with 5 percent NaOH, filtering, dialyzing over night in a collodion bag in running water, and precipitating with HCl. The maximum point of turbidity is between pH 4.90 and pH 4.66, determined with a quinhydrone electrode, and can be accurately determined with experience. The turbid solution is centrifuged and the mucin weighed wet, the excess of water removed by draining and taken up with absorbing paper. This method was accurate to $\pm$ 8 percent.

The mucin in plaques is derived from saliva. Patients tested with the method described above had from 0.10 to 0.37 percent of mucin in

stimulated saliva. Other proteins in saliva may be incorporated into plaques. Stimulated saliva contains from 1.54 to 4.62 percent by weight of a flocculent material, which settles to the bottom of a beaker when undisturbed for an hour. This mixture consists largely of proteins plus oral and salivary debris. The amount of inorganic matter in plaques, determined by ashing in a porcelain crucible, varied from 2.73 to 6.42 percent by weight. The fat in undried plaques was extracted with alcohol and ether on a steam-bath for six hours. This method gave 1.21 percent of total lipid in dental plaques.

The role of mucin in plaque formation is important. Mucin favors food stasis, and binds the mass closely to the teeth. Plaques so formed prevent oral contents from diluting or neutralizing acids in contact with enamel, and dental caries results.

DISCUSSION

Organic acids produced by fermenting carbohydrates can rapidly destroy dental enamel. An environment favorable for production of acids in sufficient concentration appears to be the primary cause of dental caries. The localization of caries indicates that this acid condition is not equally distributed throughout the mouth. Acids to be produced in sufficient concentrations to be destructive of enamel must be protected from the oral contents. Plaque presents this favorable environment. Whether plaque is essential for the progress of decalcification has not been established, but it is apparently essential for the initial lesion of caries. The passive resistance of enamel can not be the controlling factor in immunity to dental caries. Lactic acid, in concentrations found in fermenting carbohydrate mixtures, softens human enamel in twelve hours at body temperature. The rate of enamel decalcification varies with teeth in the same mouth and in different mouths, but all enamel is destroyed by this acid. It appears that the passive resistance of enamel is a factor in controlling the rate of decalcification and, to a lesser degree, the susceptibility of teeth. Any abnormality of gross structure that permits accumulation of food debris, predisposes to dental caries.

Carbohydrates are necessary for fermentation and acid production, therefore necessary for the initiation of dental caries. This may explain in part the low index of dental caries in meat eaters, such as the

Eskimos, and the high index in the civilized races on a greatly refined carbohydrate diet. Experimental dietary programs have reduced the index of caries in children by restricting the carbohydrates in the diet. The mucin in the saliva must also be a factor in predisposing to dental caries. Mellanby, working on dogs, reports that with deficient diets hypoplastic teeth were produced, but not caries. Dogs have little or no salivary mucin, which may explain the negative results. Dental caries is, therefore, a complex process dependent upon many factors.

CONCLUSIONS

(1) Organic acids produced by fermentation of carbohydrate material decalcify all human enamel *in vitro*.

(2) The rate of acid decalcification of human enamel varies.

(3) A general acid condition of the oral cavity does not produce true dental caries, but may predispose to caries.

(4) Mucin plaques present a favorable environment for acids to accumulate and decalcify enamel.

(5) Weak alkaline salts of saliva do not pass through plaques in sufficient amounts to be important in controlling acids that produce caries.

(6) Alkaline mouth-washes are of no value in controlling acids that produce caries.

(7) Soluble carbohydrates in the mouth diffuse through plaques, and under favorable conditions are changed to acids.

(8) Plaques are practically insoluble in all solutions that can be tolerated in the oral cavity.

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ADDENDUM

The following regarding the use of the apparatus (p. 856) should be added: The separated plaques were floated in distilled water in a beaker, to wash out the acid, and were kept there until ready for use. The J-tube was filled with solution to be tested, and the upper opening sealed with liquid paraffin to prevent leakage of solution while the plaque was being placed in position. The orifice of the filled J-tube was placed under a floating plaque, and so raised out of the water that the plaque covered the orifice and extended evenly over its edges. The plaque and edge of the J-tube were dried with filter paper. Hot liquid paraffin was then applied to the tube over the edges of the plaque in such a manner that none of the paraffin passed upon the area of the plaque over the orifice. The sufficiency of the seal and plaque was tested by opening the upper end of the tube. Defective plaques were rejected at the start, and imperfect seals repaired. Hydrostatic pressures were equalized by keeping the liquids inside and outside the tube at the same level. At the end of each test, the plaque and seal were again tested as above described. The recorded results apply to successful tests only.

Some of the data in this paper were included in the report at the tenth general meeting of the International Association for Dental Research, Columbus, Ohio, March 19–20, 1932: *Journal of Dental Research*, 1932, xii, p. 455; June. The paper was read at the meeting of the Research Section of the American Dental Association, Buffalo, N. Y., September 12–16, 1932.

COMMENT ON SOME MISSTATEMENTS REGARDING THE CARNEGIE FOUNDATION'S STUDY OF DENTAL EDUCATION[1]

WILLIAM J. GIES, PH.D.

School of Medicine, Columbia University, New York City

A pamphlet recently distributed with the "compliments of the School of Dental and Oral Surgery of Columbia University,"[2] contains a long series of statements by Dr. Alfred Owre purporting to show (pp. 17–47) that the Dental Educational Council of America has been and is a "reactionary body" (p. 17); that, "whatever it professes, its activities are still 'Machiavellian,' and actually on the side of lower rather than higher standards in professional education" (p. 30); that "the leading universities are fast losing their respect for the Council" (p. 42); that the Council "should be made up of cultivated, unprejudiced men, without 'axes to grind'. . . . should be incapable of political intrigue . . . . seems interested, not in education, but in the protection and extension of the vested interests in dental education" (p. 47); etc. Since July 1, 1928, Dr. Owre has "refused to have official communication with the Council" (p. 43).[3] In the many statements that these remarks illustrate, Dr. Owre discussed only matters which, seen through prejudiced or unfair comment, are damaging to the reputation of the Council. He included none by judicial fairness that are to the Council's credit. Dr. Owre, having

[1] The expense involved in adding this paper to this issue has been paid by the author. The size of this issue and volume has been increased, without expense to the *Journal*, by the number of pages occupied by the paper, which has not displaced another manuscript.—[*Ed.*]

[2] Owre: Some phases of dental education in the United States, pp. 93. "Paper prepared on request for the First Congrès International de l'Association Stomatologique Internationale, Budapest, Sep. 2–7, 1931."

[3] Dr. Owre, regretfully widening the gulf between his superiority and dentistry's inferiority, has lately resigned his membership in the International Association for Dental Research, the American College of Dentists, and the New York Academy of Dentistry. He is not a member of the local component of the American Dental Association.

stated his case against the Council, however unjustly but to his evident satisfaction, made the following related assertions (pp. 47–48):

"It was this organization [Dental Educational Council of America] which throughout the Carnegie Survey of Dental Education [1921–1926] made most of the inspections of dental schools on which the report of [sic] the Carnegie Foundation was largely based. The director of the Survey [Gies], however, was aware of the Council's shortcomings (cf. pp. 8–10, 14, 15 above). If its activities subsequent to the Survey are also shown to have been on a low plane, the validity of the Survey's findings and recommendations is practically nullified."

The wanton disregard for truth in this statement, and the obviously intentional offensiveness of the quoted comment, require a public correction. Below, for convenience in discussion, these three successive sentences are repeated and given the numerals 1, 2, and 3.

(1) "*It was this organization* [*Dental Educational Council of America*] *which throughout the Carnegie Survey of Dental Education* [*1921–1926*] *made most of the inspections of dental schools on which the report of* [*sic*] *the Carnegie Foundation was largely based.*"

This statement is a gross misrepresentation by one who knows the whole truth. The Council did not make "most of the inspections of dental schools" in the "Carnegie Survey;" the Council made none of the "inspections" after the first of the academic years "throughout" which the "Survey" was conducted; and the "report" was based chiefly on data obtained otherwise than by "inspections" of dental schools. The Carnegie Foundation's *Bulletin No. 19* (Dental Education in the United States and Canada; Gies, 1926) contains the following acknowledgment (p. 242): "*During the academic year 1921–22*, when each of the dental schools in the United States and in Canada was visited at least once, nearly all of the members of the Council, *in many special committees* of two to four members, accompanying the writer [Gies], gave freely of their time and attention to the observations at each school in both countries, and from their wide experience and detailed knowledge made weighty contributions to an understanding of the situation" [italic not in the original]. In the same *Bulletin*, in Part VI (pp. 243–617), each "Summary" relating to a school is preceded by a line recording the dates when the school was

visited ("inspected"). In 1921–22, when the members of the Council (including Dr. Owre, then a member) coöperated in "the inspections of the dental schools," there were 46 schools in the United States and 5 in Canada. I visited each of these schools in 1921–22, and in so doing had the effective coöperation of members of the Council in each of the 51 "inspections," as above indicated. Members of the Canadian Association of Dental Faculties or of the Dominion Dental Council, or both, also coöperated in the same way and spirit in the visits to the Canadian schools. The recorded dates of the visits to the schools after May 12, 1922—when the last of the 51 schools had been "inspected" in company with members of the Council—show the facts in *table 1* relating to my own unattended supplementary visits.

TABLE 1

Number of dental schools visited, in 1922–26, by the Carnegie Foundation's representative unaccompanied by members of the Dental Educational Council of America

YEAR	UNITED STATES		CANADA		TOTAL	
	Schools	Visits	Schools	Visits	Schools	Visits
1922 (second half)	8	8	2	2	10	10
1923	16	23	0	0	16	23
1924	18	22	3	3	21	25
1925	14	16	1	1	15	17
1926	1	1	0	0	1	1
Total.....	57	70	6	6	63	76

The *table* does not include several visits in which, by request, I accompanied committees of the Council as a special adviser in relation to problems in the Council's own work; it also omits my unattended visits to the Rochester Dispensary (Oct. '21; June, '22; May, '24; Dec., '25), Rochester School of Medicine and Dentistry (Dec., '25), Army Dental School (Jan., '24; Jan., '26), and the U. S. Naval Medical College, Department of Dentistry (Jan., '26), which institutions were not on the Council's official list of dental schools. Of the 51 schools in the United States and Canada in 1921–22, 35 were visited in 1922–26 at least once, *by me unaccompanied by members of the Council*, for a total of 76 visits, or an average of more than 2 visits per school; 16 schools were *not* visited by me, after the initial "inspection"

in 1921 or 1922. Of the 16 schools that I did not revisit, in 1922–26, 6 had been discontinued, merged with universities, or rated Class C, and 2 were schools in Canada.

Table 1 shows that in addition to the 51 visits to the dental schools in 1921–22, in which members of the Council were associated, I made 76 revisits in 1922–26 in which members of the Council were not associated. This total of 76 does not include the 7 visits to the schools mentioned above that were not on the Council's official list. These facts alone reveal the misrepresentation in Sentence 1 above. Other essential data were also ignored by Dr. Owre. In *Bulletin No. 19* one may see at a glance that in Part VI (pp. 243–617) the detailed financial records for each school represent the years 1920–24, inclusive; also that the miscellaneous data in Part VI extend over the year 1924–25. On page 246 it is stated: "Part VI in its entirety applies to conditions during the academic year 1924–25, except as otherwise noted." On page 638 of the lengthy Appendix appears the following: "The text of Part VI [pp. 243–617] is based on conditions in the dental schools in the United States and Canada *at the end of the academic year 1924–25*, but the 'summaries' and 'general comment' frequently extend its scope *to the end of the first half of 1925–26*. Supplementary references to *important new conditions during the second half of 1925–26, and before December 10, 1926*, are indicated in the following brief statements, which, like the data in Section A, are grouped in the alphabetic order of the abbreviated names of the schools" [italic not in the original]. The "supplementary references" to conditions, *down to December 10, 1926*, extend from pages 638 to 662, inclusive. These facts—all well known to Dr. Owre—show that for more than four years after the completion of "the inspections made by the Council," the work of the "Carnegie Survey" was continually in progress. I gave it whole-time attention with the aid of all concerned, including Dr. Owre as Dean of the Dental School of the University of Minnesota. He and the deans of all the dental schools knew that my report to the Carnegie Foundation, as of December 10, 1926, was an expression of the entire whole-time endeavor for six calendar years—from 1921 to 1926, inclusive.

Dr. Owre's irresponsible statement that the Council "made most of the inspections of dental schools on which the report of [sic] the

Carnegie Foundation was largely based" misrepresents the situation also by disregarding the fact that the said inspections were made by me in person for the Carnegie Foundation with the invited coöperation of the Council (of which Dr. Owre was a member). The Council's coöperation was given very generously and very effectively on a plan proposed by me to the Council, and with the direct informal assistance of individual members as such, in many special groups of two to four in each series of their visits to schools. These members were often selected by me—so selected, in fact, that the largest number of members of the Council (*including Dr. Owre*) would be aided to the greatest extent in the performance of their normal functions *as members of the Council*, through "field experience" in the long series of special "inspections" in 1921–22. If Dr. Owre had said, in his pamphlet, that the Council's ratings of the United States dental schools, as made in 1922 and announced in 1923, were "largely based" on the reports *to the Council* by its members who accompanied me to the schools in 1921–22 (his own reports on eight schools included), he would not have perverted the truth, and he would have stated what is well known. The foregoing familiar facts show that Dr. Owre's assertion, in Sentence 1 above, is not only untrue but also deliberately false.

(2) "*The director* [*Gies*] *of the Survey* [*Carnegie Foundation's study of dental education*], *however, was aware of the Council's shortcomings* (*cf. pp. 8–10, 14, 15 above*)."

The pages to which Dr. Owre alluded in the above sentence are incorrect references; the Council is not mentioned, nor are any of its "shortcomings" indicated, on any of these pages. I refrain from guessing which pages in Dr. Owre's pamphlet may be the ones he intended to specify, but refer the reader to pages 102–114 of the Carnegie Foundation's *Bulletin No. 19*, on dental education, where it may be seen that I was aware not only of the Council's "shortcomings" *but also of the Council's many creditable achievements*, and endeavored accurately to weigh both merits and demerits, and to state them fairly and constructively. Among the "shortcomings" of the Council to which I alluded was its failure to apply, to several *proprietary* schools, its own public definition of the grade of C for a dental school (p. 108). In Part VI of the *Bulletin*, I illustrated this situation by referring to one of these schools as follows:

"The Council's inconsistency [in giving this *proprietary* school a Class B rating instead of the Class C rating that the Council's rule required for proprietary schools] seems to have been an expression of partiality to a number of the stockholders and managers of the School—men who are *widely esteemed both personally and professionally*, and who have held or now occupy influential positions in national dental organizations. . . . . The Council's failure either to postpone a public rating while it gave this School a suitable opportunity to become acceptable by reorganization on a non-proprietary basis, as had been done effectually for other schools, or to grade the School Class C, indicated a regrettable degree of submission of a judicial function to extraneous considerations, and seemed to justify prevalent doubts as to the Council's ability to perform all of its public functions impersonally and with educational sincerity. *That the Council has encountered exceptional obstacles in the way of its purpose to give . . . . [this School] its true rating must be conceded*, but the situation clearly reveals the potency of some of the surviving commercial influences in dental education" [1923]. [Italic not in the original.]

Dr. Owre, in his pamphlet, makes much of such "shortcomings" as evidence of the Council's "machiavellian activities," on "the side of lower rather than higher standards in professional education," in behalf of "vested interests," etc. But all who know the relevant facts also know that Dr. Owre's statements are inherently insincere—that he does not "come into court with clean hands." When he was a member of the Council (1921–23), and had an opportunity courageously to apply his personal convictions and the Council's "published principles" to the proprietary school mentioned in the above quotation, he, acting on his own free judgment, gave this School a mathematical rating of 63 on a detailed point-system scale of 100. He thus individually gave this School 13 points *above* the line that separated "acceptable" schools from unacceptable ones. Later, at the same meeting of the Council, after each member's mark for the School had been recorded and the average calculated, Dr. Owre formally voted[4]

[4] During the progress of the Carnegie Foundation's study of dental education, I was privileged to attend all the meetings of the Council, and to file copies of the records. No restriction was placed upon my use of these records in the public interest. The above statement about Dr. Owre's vote is based upon my direct observations, and upon the official record, which, *in the interest of fairness to all concerned*, I now use on my own responsibility, without consulting the Council.

to rate this proprietary school Class B; that is to say, as "acceptable," in total disregard of the Council's "published principles." Probably Dr. Owre voted as he did for the reasons that obviously impelled the Council as a body to refrain from rating this School Class C—not for machiavellian or unworthy ends, but merely because the special *practical* considerations in favor of Class B outweighed the *logical* and *theoretical* factors in favor of Class C. This was a very "human" decision, and of a kind that is not at all peculiar to the Dental Educational Council of America. Dr. Owre is now intolerant of this "shortcoming" in others, but is so tolerant of it in himself that he appears to have forgotten all about the part he played.

(3) "*If its* [*the Council's*] *activities subsequent to the Survey* [*Carnegie Foundation's study of dental education*] *are also shown to have been on a low plane, the validity of the Survey's findings and recommendations is practically nullified.*"

Here both the purpose of the misrepresentations and the technique are revealed together. It was indicated in the discussion of Sentence 1 why this "three-shell game" of Dr. Owre's should not catch any "suckers." The Council did *not* make "most of the inspections of dental schools," nor did it present most of the data otherwise, on which my report to the Carnegie Foundation was based. But even if Sentence 1 were not a purposed untruth, why would or should the Council's activities, if "on a low plane" *subsequent* to the *completion* of "the Survey" [1926], automatically have any *nullifying* effect—or any other effect—on the *validity* of anything in my report to the Carnegie Foundation? There is neither logic nor common sense, but plenty of malice, in Sentence 3.

In remarks following Sentence 3 in the pamphlet, Dr. Owre expresses the opinion that the Carnegie Foundation's study of dental education—that is to say, my conduct of and report on it—did not amount to much. Dr. Owre's opinion may be wholly correct. The broadest and most fundamental phases of this study for the Carnegie Foundation were my conclusions that dentistry is (*a*) a natural division of health service ("medicine"), which (*b*) should be continued as a separately organized profession, and (*c*) its service made the *equivalent* of an oral specialty of medical practice. To further what I regard as a normal *evolution*, in the public interest, I proposed (*d*) the "two-three-*graduate* plan" of dental education, (*e*) the organi-

zation of the American Association of Dental Schools, (*f*) the effective reorganization and support of the Dental Educational Council, etc. Any one who does not endorse these views and aims—Dr. Owre does not—is just as free to reject them as I am to adhere to them. Dr. Owre seems to believe that the most effective way to prevent attainment of the main objectives outlined above, and to accomplish what he prefers, is to discredit my report to the Carnegie Foundation, misrepresent the Dental Educational Council, disorganize the American Association of Dental Schools, disparage the "two-three-*graduate* plan," disrupt and degrade the dental profession, create an "American Board of Stomatology," and get the "master-servant (1–20) plan" into operation. Dr. Owre has concluded that the recommendations in my report to the Carnegie Foundation, and my special efforts to promote the *Journal of Dental Research* and the International Association for Dental Research, tend to unify, improve, and sustain dentistry as a separately organized profession. He would prefer to nullify these influences so that he, and the few who are coöperating with him in his political intrigues, might disorganize, divide, and destroy the dental profession—and create a type of dental practice that neither dentistry nor medicine wants, and which the public could not be forced to accept. Since no alleged facts were presented by Dr. Owre to support his opinions regarding the quality of my report to the Carnegie Foundation, further comment by me on this phase of the situation would be inappropriate. But one matter in a quotation that Dr. Owre misuses in this connection calls for special attention.

On the three pages following that on which Sentences 1–3 appear, where the uselessness of my report to the Carnegie Foundation (1926) is the general theme, certain activities and relationships of Dr. Owre's are indicated (the "American Board of Stomatology" among them) as important elements in a "reawakening." Included in this revelation—and as bearing on the imperfections of dental schools, on the inefficiency, stolidity, and cupidity of the Dental Educational Council, on the ineptitude not to say harmfulness of my report to the Carnegie Foundation, etc.—is a quotation from a "*discussion not yet published,*" in which it is alleged that Dr. F. B. Noyes said that "a man very closely associated with a dental educator told me [Noyes] within a month [of December 1, 1930] that the latter filled in his income tax report last year [1929], a net profit of over $100,000 derived from a

proprietary educational institution in dentistry." (Here we are told that an intimate friend of an intimate friend whispered to an intimate friend, etc.) The contexts of Dr. Owre's remarks and of the quoted comment by Dr. Noyes indicate that United States and Canadian dental schools were reviewed, and the "net profit of over $100,000" was derived from the conduct of one dental school—"dental school" in the sense in which the term is commonly used, for otherwise the "profit" would have had no relation to the subject under consideration.[5] This "unpublished" statement by Dr. Noyes was also quoted by Dr. Owre in a discussion (February, 1931) of a paper by Dr. F. M. Casto, as published in the issue of the *Journal of Dental Research* for June, 1931 (p. 397).[6] On that occasion, in a reply to Dr. Owre's statements in this relation, I commented in part as follows (pp. 411–412):

"Dr. Owre said he approved the assertion that an unnamed somebody said to Dr. Noyes that 'a dental educator . . . . filled in his income tax report last year a net profit of over $100,000 derived from a proprietary educational institution in dentistry' (p. 397). *Dr. Owre would naturally not present this assertion as his own unless he knew it was true, and of course he knows the name of the man who made this profit.* All of the five independent dental schools have been rechartered as philanthropic institutions and, being non-proprietary, no one can legally derive private profit from the management of any of them. If any officer made $100,000 for himself 'last year' in the conduct of any dental school in the United States, he has done so illegally and under false pretenses. *I ask Dr. Owre to give us, before he leaves the meeting* [*New York Academy of Dentistry*], *the name of this crook*, so that he and the illegal conditions in the school may be effectively exposed. *An anonymous statement having an import so serious as this charge should not be accepted until adequate evidence is presented. Why should not Dr. Owre bravely reveal the facts?* [Italic not in the original.] Personally I do not believe that the 'unnamed somebody' told the truth to Dr. Noyes.

[5] The manuscript of the present paper was sent to the printer before the issuance of the September number of the *Journal of the American Dental Association*, which (xix, p. 1614) presents Dr. Noyes' discussion (as of Dec. 1, *1931*, instead of *1930*). There it will be seen that Dr. Noyes' allusion to the alleged profit of "over $100,000" was made in contradiction of a statement, by Dr. F. R. Henshaw, that *of the forty-three dental schools in Canada and the United States "not a single one is now proprietary."*

[6] Dr. Owre's annual report for 1931, to the President of Columbia University, also quoted this allegation of a profit of "over $100,000" in one of the forty-three dental schools in Canada and the United States. *Columbia University Bulletin of Information*, 1932, xxxii (no. 38), p. 377; June 18.

Why that 'unnamed somebody' did not make the profit $1,000,000 instead of merely $100,000 I cannot imagine. [Dr. Owre did not respond to the request to give the name of the 'crook' who is reputed to have taken a profit of over $100,000 'last year' from a dental school in the United States. If any responsible person can show that this has been done, this *Journal* will publicly pay to that person $1,000 for the proof, and will promptly publish the certified facts.—*Ed.*]"

No one has yet come forward with the evidence for which the *Journal of Dental Research*, for the privilege to publish the facts, would pay $1,000. Sixteen months have passed since the reward of $1,000 was publicly offered. Private information indicates unmistakably that *the alleged profit was not made in the conduct of any of the United States dental schools*, and that the allegation as it stands has no honorable place in a discussion of conditions in the dental schools, nor of the quality of the service of the Dental Educational Council or of the American Association of Dental Schools. There are well established facts to the effect that the person referred to by Dr. Noyes was associated with a proprietary course or courses *for practising dentists*—"post-graduate" courses of a kind, outside of the recognized dental schools, that neither the Dental Educational Council nor the American Association of Dental Schools has ever approved. Although Dr. Owre cannot be ignorant of these facts, he has industriously created the erroneous impression that the person referred to by Dr. Noyes was an officer in one of the dental schools. In this sorry performance, which illustrates the adage that "a partial truth may be the meanest kind of lie," there was repeated use of Dr. Noyes' *unpublished discussion* to bear false witness against dental schools, dental education, and by implication the agencies for their advancement. When directly challenged at a meeting of a professional society to substantiate the charge, Dr. Owre ran away from the obligation to reveal the whole truth about the alleged profit of over $100,000. The pages of this *Journal* are open to Dr. Owre to expose the guilty dental school. There will be paid to Dr. Owre, or to any cause he may designate, $1,000, if he presents to the *Journal*, for publication, proof of the charge he has made (behind Dr. Noyes as a shield) against the administration of an unnamed dental school in the United States. A copy of this paper has been sent to Dr. Owre with a letter calling his attention particularly to this concluding paragraph.

Quotations on the Status of Dentistry

The greatly improved standing of the dental profession among the professions . . . . is one of the most striking changes in public opinion that I have witnessed during my seventy years of observation of educational progress. . . . . I do not think I have seen during my seventy years of observation of the professions and the means of training them any change so great as that which has taken place in regard to the dental profession, and to the means of training dentists.—*Charles W. Eliot, President Emeritus of Harvard University*, on "The progress of dentistry and of the dental profession." *Address before the Dental School Society of the Phillips Brooks House Association, Harvard University, December 8, 1924; Harvard Dental School, 1925, pp. 14 and 19.*

I think it unlikely that physicians, unless they have a particular interest in the border subjects between medicine and dentistry, will maintain a permanent interest in dental research. This interest may be fostered for a period of special grants, but after these grants are used up or the individual matures, he will probably revert to medicine and not be a permanent investment for dentistry. . . . . The nucleus of . . . . [the] research group [for the advancement of dentistry] in my opinion must be dentists who have had the dental training and have a permanent interest in this field, who have taken the time to get additional training in some of the preclinical sciences. This of course corresponds to the make-up of the preclinical science group in medicine today. I feel a strong conviction, therefore, that individuals who would help dentistry should first help to develop research workers with dental training and dental interest.—*G. H. Whipple, A.B., M.D., M.A., Sc.D., Dean and Professor of Pathology, School of Medicine and Dentistry, University of Rochester; New York Journal of Dentistry*, 1931, i, p. 159; April.

Nothing is to be gained by converting a first-class dentist into a mediocre medical practitioner. . . . . I do not believe it is practical to unify the dental and medical curricula so as to provide for the same training of dentists and physicians during any considerable part of their academic careers. . . . . Dentistry is a very specialized field of medicine which has already been elevated to a standard of professional excellence entirely comparable with that of other medical specialties. *The average American dentist is at least as well qualified to render adequate service in his field of work as is the average otolaryngologist, ophthalmologist, or surgeon* [italic not in the original]. Dental education in America has been notably effective, and that is to the credit of the profession. . . . . Changes should not be made at the expense of that which has been found good through long practical experience. . . . . I am . . . . entirely unconvinced that any plan for the unification of the dental and medical curricula would result in the training of better qualified dentists. Dentistry is a profession which has become firmly established in its own right, and one which comprises a number of specialties. *It would lose much by becoming a minor subdivision of medicine* [italic not in the original]. It had best retain its independence.—*W. McKim Marriott, M.D., Dean and Professor of Pediatrics, Medical School, Washington University, St. Louis, Mo.; Journal of Dental Research*, 1931, xi, p. 807; October.

Medicine and medical education have troubles of their own which will not be alleviated by attempting to swallow whole another profession; a profession, which, by and large, does not want to be engulfed. And a profession like dentistry, with a hundred-year history, deserves to have its feelings considered. You may call dentistry a specialty of medicine, but that does not make it so; and it is none the less valuable for that fact. I can see no reason why the degree, Doctor of Dental Surgery, honestly held and honorably upheld, should not in the future deserve equal respect and esteem from the public with the degree of Doctor of Medicine, provided it is held by a similar type of man. And whatever their future relationship may be, let medicine and dentistry now go forward side by side as friendly collaborators in the estimable undertaking of offering help to those who need it and adding to the happiness of life as much as to the length of its span.—*F. T. Van Beuren, Jr., A.B., M.D., Associate Dean, and Associate Professor of Surgery, Columbia University College of Physicians and Surgeons, New York City; Journal of Dental Research*, 1932, xii, p. 249; April.

Emphatically, dentistry is a branch and not a mere twig growing on that of medicine, and I contend that the attempt to make it so is responsible for the miserably low

(*Continued on p. 4 of the cover*)

Quotations on the Status of Dentistry (continued)

standard of dentistry in this country [England]. The dental profession is . . . . lacking in self-reliance after all these years of subservience and dependence, and is like a flock of bewildered sheep, harassed on every side by conflicting interests. . . . . America the world over holds the premier position in dentistry. . . . . Can you think of a single article of modern surgery equipment which did not originate in the States, and where do you have to go if you want the very best? . . . . Why is it that in dental affairs England is so far behind America? It seems to me that the reason lies in the control of dentistry by the medical profession. . . . . To adopt a railroad simile, dentistry [in England] has got on to the wrong line. Instead of a dental train controlled by a dental staff, running on its own lines, the dental profession is relegated to a van at the tail-end of the medical train, with practically no choice as to its destination.—*H. C. Visick, L.D.S., R.C.S. Eng.: Journal of Dental Research*, 1932, xii, p. 585; Aug.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Meetings in 1931–32

SECTIONS. *Ann Arbor:* November 2. *Boston:* December 21, February 11, May 23. *Chicago:* October 9, January 17, March 11, May 20. *Cleveland:* December 14. *Columbus:* April 29. *Halifax:* October 2, November 27, January 29. *London:* January 20, March 9. *Louisville:* April 19. *Minnesota:* November 30, January 25, February 29, March 28, April 25. *New Haven:* December 7, February 17. *New York:* December 10, May 10. *Philadelphia:* November 24, March 4, April 15, June 3. *Pittsburgh:* October 5, November 2, December 7, January 4, February 1, March 7, April 4, April 25, June 23. *Prague:* May 3. *St. Louis:* October 13, January 12, April 12. *San Francisco:* March 9. *Toronto:* November 12, March 8, April 19. *Washington:* October 13, March 5.

ASSOCIATION. Tenth general meeting: Columbus, Ohio, March 19 and 20, 1932.

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CONTENTS

October, 1932

Prosthesis of the mouth and face. A symposium. *V. H. Kazanjian*, D.M.D., M.D., F.A.C.S., *Arthur T. Rowe*, D.D.S., and *Harry A. Young*, D.D.S. 651

Post-eruptive changes in the permanent dentition of dogs fed rachitogenic diets. *S. N. Blackberg*, Ph.D., and *J. D. Berke*, D.D.S. 695

A study of the incidence of dental caries in one thousand Chinese. *Bert G. Anderson*, D.D.S. 709

Medico-dental case records. XI. Endocarditis. A clinico-pathological study. *Bissell B. Palmer*, D.D.S., F.A.C.D., and *Malcolm W. Carr*, D.D.S. 713

Experiments on the dietary control of dental caries in children. *Ewing C. McBeath*, D.D.S., B.S., M.D. 723

Effect of meals, and the chewing of paraffin and gum, on the acid-neutralizing action of saliva. *J. C. Forbes*, M.A., Ph.D. 749

Studies of the cause and nature of dental caries. *J. J. Enright*, Ph.D., *H. E. Friesell*, D.D.S., F.A.C.D., and *M. O. Trescher*, M.S. 759

Local factors in dental caries. *Edward C. Dobbs*, D.D.S. 853

Comment on some misstatements regarding the Carnegie Foundation's study of dental education. *William J. Gies*, Ph.D. 865

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